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THE PLANT DISEASE REPORTER

Issued By

PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

AGRICULTURAL RESEARCH SERVICE

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Crops Research Division — Plant Industry Station, Beltsville, Maryland
Volume 41 — August 15, 1957 — Number 8

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OCCURRENCE OF TOMATO LATE BLIGHT AND EFFECT OF VARIOUS FUNGICIDES ON TOMATO YIELDS IN LOUISIANA[1]

J. B. Sinclair, R. T. Brown and W. J. Martin[2]

Summary

Late blight, incited by Phytophthora infestans, on tomatoes (Lycopersicon esculentum) in Louisiana has become increasingly important in the last 10 years. Experiments initiated to test various fungicides for control of this disease did not give the information desired because of the lack of natural infection in the experimental plots. It was noted, however, that under these conditions tomatoes sprayed at recommended rates with Manzate (70% manganous ethylenebis (dithiocarbamate)), Dithane Z-78 (65% zinc ethylenebis (dithiocarbamate)) and Vancide 51 ZW (75% zinc salt, 2-mercaptobenzothiazole), alone or in combinations, gave highly significantly greater total yields of marketable fruits over tomatoes sprayed with Copper A (Tetra copper calcium oxychloride) alone. Total yield increase was accompanied by a tendency for an increase in the production of U. S. Number 1 fruit and the number of cull fruit. The reason for this phenomenon could not be concluded from the present data.

OCCURRENCE OF TOMATO LATE BLIGHT

The occurrence of late blight on tomato (Lycopersicon esculentum), incited by Phytophthora infestans, was first reported with authentication in Louisiana by Person in 1946[3]. Departmental records show that this disease has been observed on Louisiana field tomatoes every year since 1946 up to and including 1954 and 1957. Tomato late blight reached epiphytotic proportions during the spring growing seasons of 1947 and 1950.

FUNGICIDE STUDIES

Experiments were started in the fall of 1950 and spring of 1951 to determine the relative effectiveness of various recommended fungicidal sprays and dusts in their ability to control late blight on Marglobe tomatoes under Louisiana conditions. The treatments were replicated four times in completely randomized plots located at either the Louisiana State University Hill Farm at Baton Rouge, or the Barry Farm at Prairieville. The recommended horticultural practices for this area were followed using DDD (Rothane) for insect control. The 1950 Hill Farm experiment had 20-foot plots with 10 plants per plot; the 1950 Barry Farm experiment had 40-foot plots with 20 plants per plot; and the 1951 Barry Farm experiment had 100-foot plots with 50 plants per plot. Records of the total marketable fruits from treated and untreated plots were taken and field observations of disease occurrence were made. No late blight was recorded in any of the experiments during the seasons in which they were grown. Phoma leaf spot, incited by Phoma destructiva, was found in both experiments of the 1950 planting. On nonsprayed plants this disease caused more damage than on the treated plants, which might account for some of the differences in yields. There was slight infection of Cladosporium fulvum recorded in the 1950 Hill Farm Experiment. Early blight, incited by Alternaria solani, was present in all plots, both on treated and nontreated plants in the experiment made in the spring of 1951.

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[1] Appreciation is extended to the Rohm and Haas Chemical Company for providing Dithane D-14, and Dithane Z-78; to the Carbide and Carbon Chemical Company for Potato Fungicide 658; to the Niagara Chemical Company for Vancide 51 ZW; the E. I. DuPont de Nemours and Company, Inc. for Manzate and Copper A; the Tennessee Copper Company for Tri-basic copper sulfate and the Stauffer Chemical Company for Captan.

[2] Assistant Plant Pathologist, Superintendent of the Plaquemines Parish Experiment Station and Plant Pathologist, respectively, of the Louisiana State University and Agricultural and Mechanical College, Agricultural Experiment Station, Baton Rouge, Louisiana.

[3] Person, L. H. 1946. Late blight on potato and tomato in Louisiana. Plant Dis. Reptr. Suppl. 164: 274.

Table 1. Treatment means of total yield in pounds per acre of Marglobe tomato for plot location and year indicated.

Treatment and Rate	Plot Location and Year		
	Hill Farm Fall 1950	Barry Farm Fall 1950	Barry Farm Spring 1951
Tribasic copper sulfate 4 lbs./100 gals. water	3,021	6,698	17,696
Potato Fungicide 658 3 lbs./100 gals. water	1,582	8,113	—[a]
Dithane Z-78 1.5 lbs./100 gals. water	3,275	8,527	19,254
Tribasic copper sulfate 7 per cent dust	2,390	6,147	18,557
Potato Fungicide 658 6 per cent dust	3,115	7,811	—[a]
Dithane Z-78 6 per cent dust	3,618	7,460	18,883
Non-treated	2,127	5,678	16,258
L.S.D. 5%	N.S.	N.S.	N.S.

[a] Treatment not applied.

Table 2. Treatment means for total yield in pounds per acre of Moreton hybrid tomatoes for class indicated, Spring 1954.

Treatment and Rate in lbs./100 gals. Water	Marketable Fruits	U. S. No. 1 Fruits	Cull Fruits
Vancide - 2	23,191**	8,710	19,329
Dithane Z-78 - 2	26,002**	10,071	20,034
Mansate - 2	24,879**	11,106	16,550
Copper A - 4	18,673	8,656	17,421
L.S.D. 5%	1,851	N.S.	N.S.
1%	2,450	N.S.	N.S.

Table 3. Treatment means for total yield in pounds per acre of Moreton hybrid tomatoes for class indicated, Spring 1955.

Treatment and Rate in lbs./100 gals. water	Marketable Fruits	U.S. No. 1 Fruits	Cull Fruits
Vancide - 2	29,125	9,962	29,615*
Dithane Z-78 - 2	33,862**	11,487	27,274
Manzate - 2	33,154**	10,452	32,120**
Copper A - 2	31,194*	11,868*	28,527
Copper A - 2 plus Manzate - 1	33,971**	11,759*	27,655
Manzate - 1 plus Dithane Z-78 - 1	32,501**	8,329	28,254
Captan - 2	30,541	9,908	30,323**
Copper A - 4	29,343	9,527	25,968
L.S.D. 5%	1,524	2,069	2,831
1%	2,069	N.S.	3,865

The data for the three experiments were adjusted to yield in pounds per acre. The statistical analysis of these data showed no significant differences between the treatments and the check (Table 1). The lack of significance possibly was due to the wide variability in the replications, part of which may have been due to pilfering and the presence of the other foliage diseases. It was noted that there was a tendency for higher yields from plots treated with Dithane Z-78 (65% zinc ethylenebis (dithiocarbamate)) and Potato Fungicide 658 (copper zinc chromate complex) than plots treated with 53% tri-basic copper sulfate at the rates used. It was thought that this observation warranted further testing under more controlled conditions.

In the spring of 1954 and 1955 a series of similar experiments using spray treatments were carried out at the Plaquemines Parish Experiment Station. The variety Moreton hybrid was replicated three times in 20-foot plots with 10 plants per plot and the fungicide treatments were completely randomized within each replicate. The recommended horticultural practices were followed and insect pest control was used consistently. Records of the yield of marketable and cull fruits were taken as well as the yield of U. S. No. 1 tomato fruits. These data were adjusted to total yield in pounds per acre.

In 1954, the effects of four fungicide spray treatments on the yield of the Moreton hybrid tomato were compared. Vancide 51 ZW (75% zinc salt, 2-mercaptobenzothiazole), Manzate (70% manganous ethylenebis (dithiocarbamate)), and Dithane Z-78 were applied at a rate of 2 pounds per 100 gallons of water and Copper A at a rate of 4 pounds per 100 gallons of water as recommended by the manufacturers of these fungicides. Yield data in pounds per acre were statistically analyzed (Table 2). Plants sprayed with the three organic fungicides showed a highly significant difference in yield of marketable fruits over plants sprayed with Copper A (Tetra copper calcium oxychloride). There were no significant differences between treatments in yield of U. S. No. 1 fruits or in the number of cull fruits, although there was a tendency for the yield of U. S. No. 1 fruits to be higher for the organic sprays than for Copper A. Vancide and Dithane Z-78 tended to have more culls and Manzate less culls when compared with Copper A.

The 1954 experiment was repeated in 1955, but expanded to include Captan at 2 pounds per 100 gallons of water, Copper A at 2 pounds per 100 gallons of water, a combination of Manzate at 1 pound per 100 gallons of water plus Copper A at 2 pounds per 100 gallons of water, and a mixture of Manzate at 1 pound per 100 gallons of water plus Dithane Z-78 at the same rate. When the total marketable fruits of each treatment is compared with Copper A at

4 pounds per 100 gallons of water all except Vancide, Captan and Copper A at 2 pounds per 100 gallons of water show a highly significant increase (Table 3). Even though all treatments tended to yield more U. S. No. 1 fruits (except the Manzate-Dithane Z-78 combination) only Copper A at 2 pounds per 100 gallons of water and the Copper A-Manzate combination showed a significant increase. All treatments tended to have more culls than Copper A at 4 pounds per 100 gallons of water, with Manzate and Captan being highly significantly greater and Vancide showing a significant increase.

When the rate of Copper A was reduced from 4 to 2 pounds per 100 gallons of water a significantly higher yield of marketable and U. S. No. 1 fruits was found, but no significant difference was found in the number of culls. Copper A at 2 pounds per 100 gallons of water combined with Manzate gave a highly significantly greater yield of marketable fruits, a significantly higher yield of U. S. No. 1 fruit and no increase in cull fruit over Copper A at 4 pounds per 100 gallons of water.

No late blight was recorded at or in the area of the Plaquemines Parish Experiment Station during the springs of 1954 and 1955, but early blight, incited by Alternaria solani, was present during both growing seasons. Since most of the fungicides used gave good control of this disease, it was not considered important.

DISCUSSION AND CONCLUSIONS

In the absence of late blight infection on Louisiana spring tomatoes the use of various organic fungicides recommended for control of this disease gave a significantly higher yield of marketable fruits when compared with Copper A, at the rates used. A tendency for an increase in U. S. No. 1 fruits and the number of culls was noted in plots sprayed with the organic fungicides. Whether this difference would be marked under severe late blight conditions is not known.

The presence of early blight and its possible effect on yields was not considered important since all the fungicides used gave good control of this disease.

It might appear from these data that Copper A was phytotoxic, but this cannot be concluded with certainty.

LOUISIANA STATE UNIVERSITY AND AGRICULTURAL AND MECHANICAL COLLEGE, AGRICULTURAL EXPERIMENT STATION, BATON ROUGE, LOUISIANA, AND PLAQUEMINES PARISH EXPERIMENT STATION

TESTING SWEETPOTATOES FOR BLACK ROT RESISTANCE

E. M. Hildebrand[1, 2]

Summary

The reported study demonstrated the value of preliminary experiments for designing methods capable of giving consistent results, and the feasibility of determining the status of black rot resistance of 300 sweetpotato seedling selections or unknowns.

In the preliminary phase, methods, which are described, were developed for production of inoculum, preparation of roots for inoculation, inoculation of roots, incubation in a controlled environment, and recording of results. The methods were standardized to ensure dependable results in successive tests over periods of months.

Three virulent cultures of the black rot fungus, from Beltsville, Maryland, the University of Maryland, and Louisiana State University, were used throughout. Two known resistant and susceptible check varieties recurred in all experiments. Every clone or lot tested consisted of three average size roots. Each root was inoculated at six points with one of the three cultures and incubated in moist chambers at 21° C for 3 weeks.

Data were recorded on diameter of surface lesion and on estimated area of lesion surface exposed by a transverse cut through the root at point of inoculation.

The check varieties showed consistent and highly significant differences in response to inoculation by all three cultures, according to both criteria.

The Beltsville culture produced significantly larger surface and internal lesions on the susceptible check than did the other cultures. The Louisiana culture produced only slightly larger surface and internal lesions on the resistant check than did the Beltsville culture but significantly larger ones than did the Maryland culture.

The two criteria for measuring response to inoculation showed equally consistent results as measured by coefficient of variation, but relative differences in values for internal lesions of the resistant and susceptible checks were greater than for surface lesions.

The frequency of ratings of the 300 unknown clones showed an approximately normal distribution. The distribution for both cross-section area and average surface diameter suggested a bimodal tendency, but this tendency was less marked for the latter. By both criteria a slight skewness toward resistance was evident.

A few clones showed significantly higher resistance than the resistant check, and a few significantly higher susceptibility than the susceptible check, as measured by lesion diameter.

In response to inoculation, aging differences occurred within varieties among successive tests over periods of several months, showing the necessity for standard checks in every experiment. In evaluation of varietal resistance from the market standpoint, surface diameter rather than internal area of lesion appears the more practical criterion.

Black rot, caused by Ceratocystis fimbriata Ell. & Halst., is still one of the most destructive and widespread diseases of sweetpotatoes (Ipomoea batatas (L.) Lam.) in the United States. The causal fungus attacks the roots and sprouts in the plant bed and in the field, and also the roots in transit and storage. For many years control measures were largely directed toward the use of chemicals and of heat treatments to reduce losses since all common varieties of sweetpotato were susceptible (2, 3, 6, 7, 9, 10, 11, 12).

In 1953 Cheo (1) demonstrated that a few of the sweetpotato varieties and seedlings that he tested were highly resistant to black rot. Martin (8) reported in 1954 that the reaction of a

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[2] The author is indebted for statistical analyses to E. James Koch, Biometrical Services, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

variety or seedling may depend on the specific parasitic race of the causal fungus to which it is exposed.

The study reported herein is essentially an extension of the work of Cheo and of Martin, designed to obtain data on many more seedlings, potential varieties, and newly introduced varieties. The first objective was to re-examine the testing procedures and to design one suitable for evaluating root resistance or susceptibility at any time during the storage life of the root. The second objective was to evaluate a considerable number of selections or unknowns by employing two different measurements of disease severity, namely, surface diameter and cross-section area of lesions.

PRELIMINARY EXPERIMENTS

To achieve the first objective three preliminary experiments were conducted in the autumn of 1954, using roots of the very susceptible Porto Rico and Orange Little Stem varieties.

The first task was to work out an inoculation procedure that could be used with reasonable certainty for testing the resistance of unknowns at any time during the year. The physical limitations on numbers were rather severe since only 20 lots could be tested at one time and 3 weeks were required to complete one experiment. The second requisite was to determine whether there was any change in the status of root resistance within varieties at different times during the year. To accomplish this purpose certain known resistant and susceptible varieties were used as controls in each experiment.

The three preliminary experiments, begun in October and finished in December, were successful in the standardization of the steps of the procedure, which are described as follows:

1. Preparation of Roots: Root selection, examination, and preparation constituted the first step, because it was necessary to start with disease-free roots in evaluating them for black-rot resistance. The first examination was made before removal from storage. Each lot consisting of three selected roots was washed in running water and spread on paper at room temperature to dry, then dipped in bichloride of mercury (1-1000) and dried as before. When completely dry each root was labeled with a wax pencil, 1) to identify the inoculum to be used and 2) to locate with small circles the six points on each root to be wounded and inoculated later.

2. Kind and Quantity of Inoculum: Sporulating mycelium of the black rot fungus was used in preference to spore suspensions because its preparation and use were easier to standardize (4). Inoculum was obtained from cornmeal agar cultures incubated 2 to 4 weeks at 26° C in 150 ml Erlenmeyer flasks. Thin strips of agar bearing mycelium were transferred, with a thin spatula, bent into an L, from the flask to a Petri dish and then cut into 2-mm cubes with a scalpel.

3. Kind and Number of Wounds: A blunt flamed probe needle 1 mm in diameter was used for producing root wounds to a depth of 8 mm, since size (0. 1 mm; 1. 0 mm; 2 mm or above) made no difference in amount of infection when the needle penetrated to more than 5 mm in depth. Holding the root proximal end up, six wounds were made in two longitudinal rows about 2 inches apart in the positions previously ringed with a wax pencil before the root was placed in the moist chamber. The wounds were spaced in pairs in the proximal (1/4), medial (1/2), and distal (3/4) regions down the length of the root, equidistant from each other. Three wound punctures were made in the first row, the root was rotated about 60 degrees, and the process repeated for the second row. In the figures these three positions are indicated by the symbols P, M, and D.

4. Inoculating the Wounds: With a tiny flamed scalpel a cube of the inoculum was transplanted to the mouth of each wound in the order in which the wounds were made (left, top to bottom; right, top to bottom). Next, with the probe, the inoculum was pushed into the wound and to the bottom in the same sequence. The inoculated roots were immediately placed in the moist chambers, which were transferred to the incubators.

5. Temperature and Length of Incubation Period: To maintain optimal conditions for both parasite and host, the inoculated test roots were incubated at 21° C for 21 days. Every experiment also included a pair of checks, consisting of three inoculated roots in each of two moist chambers held in incubators maintained, respectively, at about 6° below and 6° above

the optimum of 21° C. Being held below and above the optimum temperature for infection, the check roots always developed less black rot than roots at the optimum. Efficiency of the inoculation procedure and adequacy of the inoculum were evident at all three temperatures.

6. Control of Variation due to Increasing Age of Roots: Even though roots were stored under ideal conditions (55° F at high humidity), the progressive aging of successive lots before testing constituted a variable which was controlled successfully by incorporating into each experiment two checks consisting of known resistant and susceptible varieties of the same age as the unknowns or test lots.

7. Humidity Maintenance and Contamination Control: The use of a disinfectant (mercury bichloride, 1-1000) was necessary for contamination control in moist chambers, since fungus contamination was generally present on the surface of sweetpotato roots. To maintain high humidity one or two layers of filter paper moistened with disinfectant were used to cover the bottom of the dish.

8. Sources of Inoculum: Preliminary trials left no doubt of the need for more than one source of black rot inoculum: therefore the maximum of three sources was used. The 10-inch moist chambers would accommodate three average-size roots, each of which was inoculated with mycelium from a different source. For uniformity and avoidance of cross-contamination, each root was inoculated with only one fungus strain. The cultures were labeled according to source: "B" for Plant Industry Station, Beltsville; "L" for Louisiana State University; and "M" for the University of Maryland. The arrangement adopted provided six wounds on separate roots for each of the three fungus strains. It gave uniformity of response to inoculations paired crosswise but not lengthwise of the roots. Individual roots were not sufficiently large in circumference to accommodate three longitudinal rows of wounds without danger of contact of one row with the disinfectant on the bottom of the dish.

9. Recording of Results: Severity of black rot was determined at the paired wounds in three regions of the root by: 1) measurement of lesion diameter at the root surface, and 2) estimation of the areas of the lesions exposed in cross sections and/or longitudinal sections. Surface diameters of lesions were measured weekly and areas of lesions exposed in cross sections were estimated on an indexing scale at the end of the experiment.

Figure 1 illustrates the criterion of cross-sectional areas used for evaluating root resistance to black rot. The test lots were divided into four classes: 1 (A in the figures), for very resistant; 2 (B), for resistant; 3 (C), for susceptible; and 4 (D), for very susceptible. Number 1 represents a trace of infection, whereas 4 was based on actual measurements and represents 4 cm^2 or more of cross-sectional area per lesion. Numbers 2 and 3 represent visual estimates of approximately but usually less than 2 and 3 cm^2, based on a trace of infection at 1 progressing to an area of 4 cm^2 or more at 4.

The same materials were also examined in longitudinal section but these data were omitted from the report. Figure 2 illustrates one lot of the susceptible check variety, Orange Little Stem, in both cross and longitudinal section through the proximal, medial and distal regions of the roots. The three roots illustrated demonstrate the infections induced by the Beltsville, Louisiana, and Maryland cultures of the black rot fungus when incubated at 21° C. The longitudinal sections were made from essentially the mirror images of the left hand lesion illustrated in cross section. It is evident that the longitudinal spread of the fungus was usually more extensive than the lateral spread, and often of a different character.

VARIETAL EVALUATION

Materials and Methods

Evaluation of sweetpotato varieties for susceptibility to black rot was begun in December 1954. It involved 15 experiments which included 200 lots during 1954-55 and 100 lots during 1955-56. The sweetpotato varieties used were part of the collection at Beltsville, Maryland, and were supplied by C. E. Steinbauer. The laborious inoculation method developed during the preliminary experiments gave readily reproducible and comparable results in all succeeding tests.

The values for surface-lesion diameter measurements in the tables and the graph are

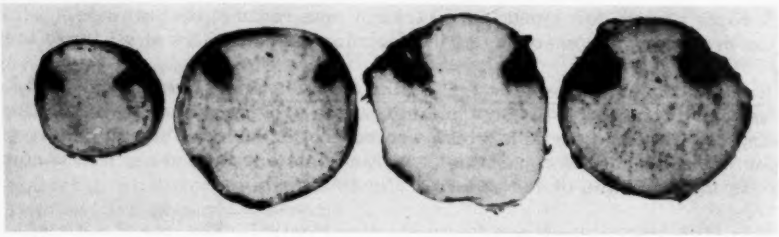


FIGURE 1. One criterion for recording sweetpotato root resistance to black rot was cross-sectional area of lesion: A for very resistant, B for resistant, C for susceptible, and D for very susceptible. Class A represents a trace of infection, whereas D represents 4 cm^2 or more, and B and C intermediate areas. x about 3/8.

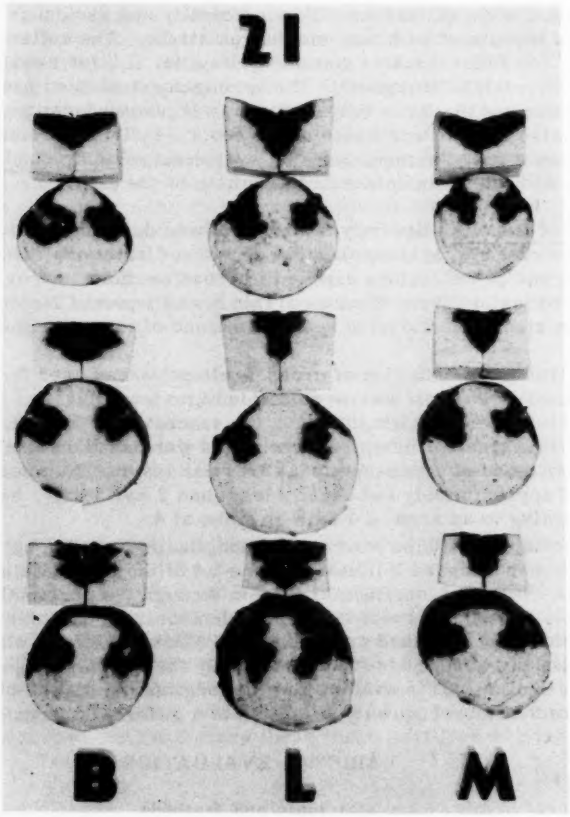
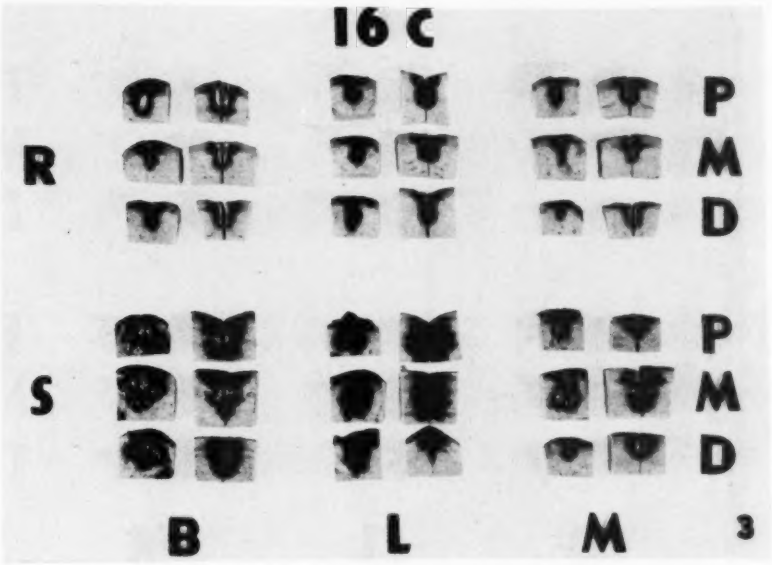


FIGURE 2. Susceptible Orange Little Stem sweetpotato in both cross and longitudinal section through black rot lesions in (top to bottom) the proximal (P), medial (M), and distal (D) regions of three roots inoculated with the (left to right) Beltsville (B), Louisiana (L), and Maryland (M) cultures, and incubated at 21° C for 3 weeks. The longitudinal sections in the upper position were made from the mirror images of the cross-sections in the left position. x about 3/8.



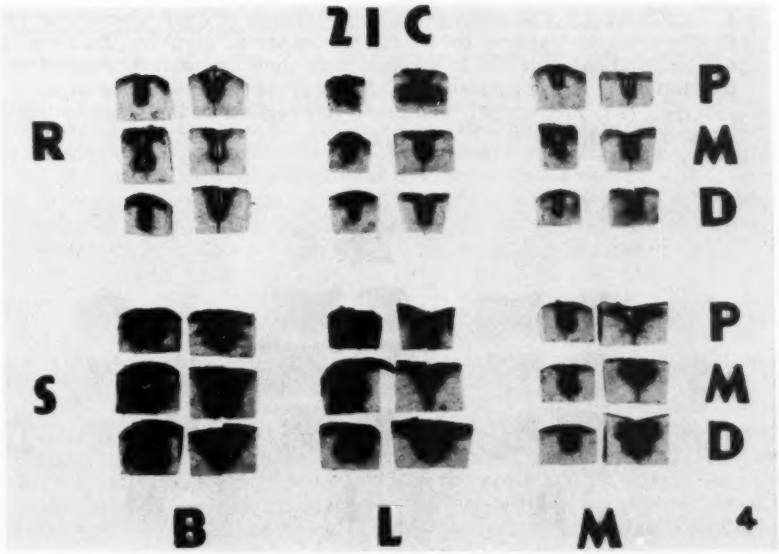


FIGURE 3, FIGURE 4. The response of the resistant (R) and susceptible (S) sweetpotato checks from the experiment concluded on April 7, 1955. The cross and longitudinal sections in juxtaposition show the lesions induced by the Beltsville (B), Louisiana (L), and Maryland (M) cultures in the proximal (P), medial (M), and distal (D) regions.

FIGURE 3.-- roots incubated at 16° C. FIGURE 4.-- roots incubated at 21° C. x about 3/8.

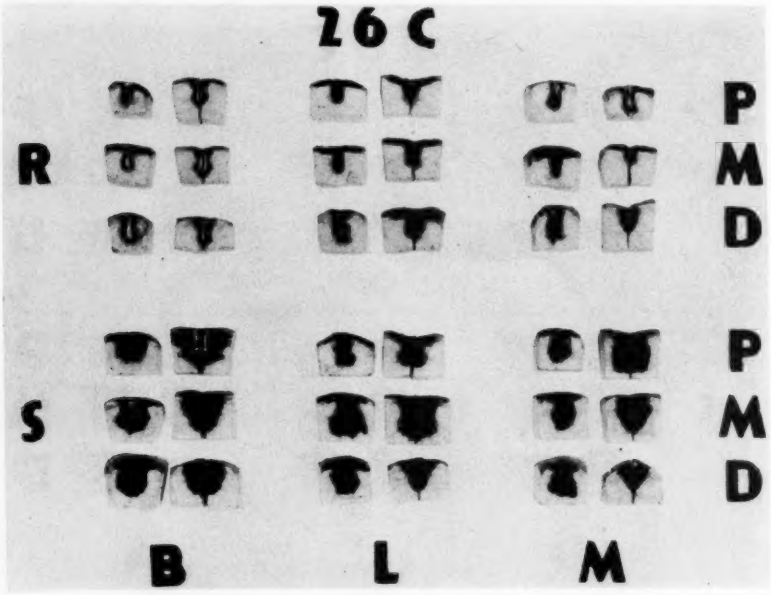


FIGURE 5. The response of the resistant (R) and susceptible (S) checks from the experiment concluded on April 7, 1955. The cross and longitudinal sections in juxtaposition show the lesions induced by the Beltsville (B), Louisiana (L), and Maryland (M) cultures in the proximal (P), medial (M), and distal (D) regions of the roots incubated at 26° C. x about 3/8.

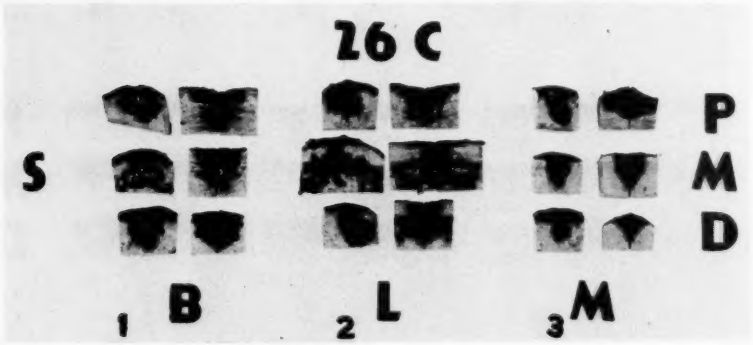


FIGURE 6. Secondary invasion of the unwounded root interior by the black rot pathogen after aging had formed cracks or open pathways for unimpeded invasion by the fungus. The illustration is of susceptible Orange Little Stem incubated at 26° C for 21 days. Note that secondary invasion is slight in root 1, marked in root 2, and absent in root 3. As to size, root 2 was largest and 3 smallest. x about 3/8.

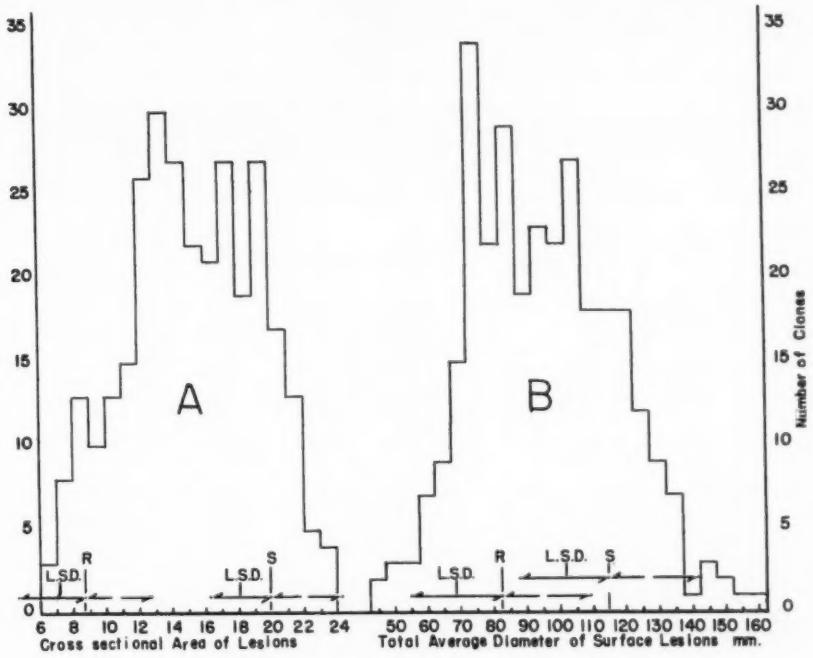


FIGURE 7. Frequency distributions for susceptibility to black rot infection of 300 unknowns. These sweetpotato clones were rated for resistance or susceptibility to the black rot fungus (averages of 3 cultures) according to two criteria for evaluation: A, mean cross-sectional area of lesions (index scale), and B, mean diameter of surface lesions (mm). The L. S. D. values on the graph were calculated for between tests of the check varieties only and are to be applied to the frequencies presented in the graph. (See Tables 1 and 2 for other L. S. D. values.)

means of the sums of measurements of six lesions per root for one fungus culture. The values for cross-sectional area estimates are means of the sums of the area estimates on six lesions per root.

Photographs made twice each year at intervals of about 3 months recorded the reactions of the resistant and susceptible checks to help insure consistency in classifying response of the seedling variety selections called the unknowns. Figures 3 (15° C), 4 (21° C), and 5 (27° C) illustrate the response to black rot inoculation in the resistant and susceptible checks from the test concluded on April 7, 1955. In general, the fungus made most progress at the optimum temperature of 21° C. The photographs show the usual variation in the extent and character of invasion, as viewed in both cross and longitudinal section.

The ratings of the unknowns evaluated in the several tests should be comparable, since all readings were made relative to the recurrent controls. This assumes that the proportionate changes in resistance associated with age were similar in the several test lots.

Experimental Results

Table 1 summarizes the sum values for surface lesion diameters on all the resistant and susceptible check roots in the 15 experiments. It will be observed that on the susceptible check roots the Beltsville culture produced significantly larger lesions than either the Louisiana or Maryland culture, which were about the same. On the resistant check, the Louisiana culture was slightly the most agressive. While the Louisiana and Beltsville strains were not significantly different from each other in virulence, both were significantly more aggressive than the

Table 1. Surface diameter of sweetpotato black rot root lesions on susceptible and resistant checks in the 15 experiments conducted under controlled conditions during the 1954-55 and 1955-56 seasons[a].

Experiments		Lesion diameter for variety and culture shown[b]							
		Susceptible variety				Resistant variety			
No.	Date begun	B	L	M	Average	B	L	M	Average
1	12-15-54	144	91	142	126	102	111	85	99
2	1- 6-55	136	123	129	129	90	136	37	88
3	1-28-55	135	113	102	117	76	91	65	77
4	2-21-55	129	113	98	113	76	108	64	83
5	3-15-55	137	153	88	126	83	93	58	78
6	4- 6-55	115	114	127	119	99	104	81	95
7	4-28-55	146	138	110	131	111	97	96	101
8	5-20-55	139	114	152	135	94	110	110	105
9	6-10-55	119	142	102	121	103	98	65	89
10	7- 1-55	109	93	103	102	74	99	83	85
11	1- 6-56	142	104	106	117	87	110	52	83
12	1-27-56	130	96	92	106	88	59	32	60
13	2-17-56	122	72	94	96	87	73	32	64
14	3- 9-56	107	82	90	93	95	74	52	74
15	3-30-56	107	87	89	94	90	52	34	59
	Mean	128	109	108	115.0	90.3	94.3	63.1	82.6

L. S. D. 5 percent. Between: varieties, 7.0; cultures within variety, 12.2; tests within variety, 27.2.

L. S. D. 1 percent. Between: varieties, 9.4; cultures within variety, 16.2; tests within variety, 36.3.

[a] The individual values are totals for 6 lesions per root. The L. S. D. 's of the distributions for surface diameter are shown above at the 5 and 1 percent levels.
[b] Based on measurement in millimeters of average diameter of lesion.

Maryland strain at the .01 probability level.

Table 2 summarizes the sum values for cross-sectional area of the black rot lesions in all the resistant and susceptible check roots. It will be observed that by this second criterion also the Beltsville culture was significantly more aggressive on the susceptible checks at the 01 level than either of the other cultures. On the resistant checks the Louisiana culture again was the most virulent of the three, and significantly more so at the .01 level than the Maryland culture.

In Tables 1 and 2 the differences among experiments in size of lesions were highly significant. This is partly due to some decline in susceptibility with aging. However, some variation in environmental and associated factors from test to test undoubtedly is partially responsible. For both surface diameter and cross-section area there appears to be a seasonal difference, since the values were less during 1955-56 than during 1954-55.

For the check lots the correlation coefficient (r) between diameter of lesion and cross-section area of lesion was 0.745, significant at the 0.01 level. The coefficient of determination (r^2) of 0.555 indicates that 55.5 percent of the variation in diameter is associated with the variation in cross-section area. The coefficient of variation for the diameter readings was 16.8 percent and for the cross-sectional areas was 16.4 percent.

Figure 7-A, illustrating the frequency distribution of the cross-section areas of lesions on the 300 unknowns, suggests some bimodal tendency. The means for all the susceptible and resistant checks are shown in the figure by S and R, respectively, together with L. S. D. values

Table 2. Cross-section area values of sweetpotato black rot root lesions for the susceptible and resistant checks in the 15 experiments conducted under controlled conditions during 1954-55 and 1955-56 seasons[a].

Experiments		Index values for variety and culture shown[b]							
		Susceptible variety				Resistant variety			
No.	Date begun	B	L	M	Average	B	L	M	Average
1	12-15-54	25.0	24.0	26.0	25.0	12.0	19.0	14.0	15.0
2	1- 6-55	27.0	26.0	25.0	26.0	8.0	12.5	5.5	8.7
3	1-28-55	22.0	20.0	19.0	20.3	9.0	10.0	10.0	9.7
4	2-21-55	23.0	18.5	19.5	20.3	6.0	9.0	6.0	7.0
5	3-15-55	21.5	19.5	12.0	18.0	8.5	12.5	7.0	9.3
6	4- 6-55	24.0	22.0	21.0	22.3	6.0	9.0	7.5	7.5
7	4-28-55	22.5	18.0	26.5	22.3	9.0	8.5	6.0	7.8
8	5-20-55	24.0	24.0	24.0	24.0	8.0	8.0	10.0	8.7
9	6-10-55	19.0	21.0	12.0	17.3	8.0	11.0	6.5	8.5
10	7- 1-55	18.0	20.0	18.0	19.0	8.5	13.0	11.5	11.0
11	1- 6-56	24.0	18.5	13.5	19.0	12.0	12.0	6.0	10.0
12	1-27-56	22.0	15.5	16.0	17.8	9.0	6.0	6.0	7.0
13	2-17-56	21.0	16.0	21.0	19.0	6.0	6.0	6.0	6.0
14	3- 9-56	18.0	13.5	15.0	15.5	9.5	7.0	6.0	7.5
15	3-30-56	24.0	15.5	21.0	20.2	9.5	7.5	6.0	7.7
	Mean	22.30	19.50	19.30	20.37	8.60	10.07	7.60	8.76

L. S. D. 5 percent. Between: varieties, 1.0; cultures within variety, 1.8; tests within variety, 3.9.

L. S. D. 1 percent. Between: varieties, 1.4; cultures within variety, 2.3; tests within variety, 5.2.

[a] The individual values are totals for 6 lesions per root. The L. S. D. 's of the distributions for cross-section area are shown above at the 5 and 1 percent levels.

[b] Based on scale of 1, equals most resistant, to 4, equals most susceptible. The 1 represents a trace of infection whereas 4 was based on actual measurements and represents 4 or more square centimeters of cross-sectional area per lesion, with 2 and 3 being intermediate.

at the 0.01 level. Some of the individual unknowns were more resistant or more susceptible than the mean of the respective checks, but not significantly so at the 0.01 level. The checks were widely separated.

Figure 7-B shows the frequency distribution of the 300 unknowns classified according to the diameters of surface lesions. The distribution is roughly normal, but somewhat skewed in the direction of resistance. It is only slightly less indicative of the bimodal distribution suggested in Figure 7-A. The checks as shown in Figure 7-B are nearer to the center of the distribution than in 7-A and are not so distinctly separated.

It appears that in the materials tested the degrees of deep internal fungus invasions of the resistant and susceptible sorts differ more sharply than the degrees of destruction of surface tissues. Therefore, for scientific evaluation, the criterion "cross-sectional area" might appear preferable to "diameter of surface lesions." However, from a market standpoint surface damage may be quite as important as total damage and therefore cannot be set aside.

DISCUSSION AND CONCLUSIONS

The black rot fungus itself is not very well understood, as the recent revision of its taxonomy shows (5). A preliminary comparison demonstrated both cultural and pathogenic vari-

ability in all of several isolates tested, thus supporting Martin's findings (8). Therefore, in the adopted procedure three strains were employed.

This study used a modification of Cheo's criterion for evaluating root resistance, in which he divided the test lots into four classes (1). The chief modification is in the designation of infections resulting in an extended invasion of the root interior, as illustrated in Figure 6. In Cheo's report his figure "F" (1, p. 49) was designated as Type IV reaction for "very susceptible." This condition occurs only late in the storage season, when aging and desiccation have produced cracks or open pathways that permit the fungus to progress freely through the root interior. All but the first, i.e., most resistant, type of reaction led to such secondary spread. In the experiments reported herein secondary spread was designated by the number 5 appended to the estimated injury value.

This study confirmed Cheo's finding (1) of high black rot resistance in a few seedlings or unknowns, but by inoculation with three different strains of the fungus it subjected the lots or clones evaluated to a more extensive and rigorous test. On the basis of Cheo's results the seedling B-5944, later named Sunnyside, was used for the resistant check, with Orange Little Stem or Yellow Jersey for the susceptible check.

Variability of the three strains in spore production made it necessary to use cubes of mycelial growth on agar as inoculum (4) instead of an unstandardized mass spore suspension (1).

Incorporation of known resistant and susceptible controls in each experiment made results reproducible at any time during the season possible. The relatively stable reaction during and between seasons of checks stored under optimum conditions for preservation, as demonstrated in the tables, indicates the reliability of this method.

It is evident that more basic work is needed on the pathogen itself, on the nature of strain variation, and on pathogenicity in relation to sporulation, geographic location, and climatic conditions.

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BACTERIAL WILT OF PINTO BEANS ON SOILS OF DIFFERENT TYPES AND CROPPING HISTORIES

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In northeastern Colorado and southwestern Nebraska, extensive loss frequently occurs in hail-injured beans infected by the bacterial wilt organism, Corynebacterium flaccumfaciens (Hedges) Dowson or C. flaccumfaciens var. aurantiacum Sch. & Chris. The disease is usually found in beans grown in coarse-textured, sandy soil. It is most extensive in fields where beans are grown repeatedly. However, it occurs frequently in fields growing beans for the first time, even though certified disease-free seed is planted.

To obtain more direct information on the influence of soil type and cropping history on the incidence of bacterial wilt, I conducted a study in pinto bean fields in the vicinity of Greeley, Colorado in 1955.

INCIDENCE OF BACTERIAL WILT ON PLANTS IN CLAY LOAM AND SANDY LOAM

A comparison of the incidence of bacterial wilt was made in 11 bean fields distributed in adjacent areas of sandy loam and clay loam. The fields varied in the number of bean crops they had borne. In each field, shortly after plant emergence, 40 plants (10 each in the vicinity of the corners of the field) were injured below the soil line by punching through the hypocotyl with a pointed steel rod to favor infection by soil-borne wilt bacteria (2). The number of injured plants developing bacterial wilt symptoms before harvest as well as the incidence of wilt in the rest of the field (nonpunched plants) was recorded. The data are summarized in Table 1.

No hail injury occurred in the fields under observation; so bacterial wilt incidence was comparatively low. However, infections apparently resulted largely from soil-borne inoculum introduced through mechanical injuries. Most of the infected plants other than those deliberately injured bore scars of cultivator injuries on below-ground portions of the hypocotyl. Disease incidence was considerably higher on punched plants in each field than on those not punched.

Bacterial wilt occurred on plants in sandy loam in all parts of each field regardless of cropping history. The highest incidence was in punched plants in the field bearing its third consecutive bean crop. On the other hand, in the finer textured clay loam, the disease exceeded a trace only in fields on which beans had been grown repeatedly and even there the incidence was low. This indicates that in the locality of the study bacterial wilt is favored by coarse-textured soil.

The general occurrence of the disease in the sandy area tends to support previous indications that the wilt pathogens are spread in wind-blown soil (1). Concurrently, the pattern of disease incidence in clay-loam fields indicates that inoculum potential is important in symptom expression and that repeated planting is necessary in clay loam before bacteria accumulate in sufficient numbers to produce wilt symptoms. It may be inferred that wilt bacteria in low concentrations infect plants and multiply in them during a season without producing disease symptoms. Such an ability has been indicated by other pathogens in the genus Corynebacterium (4, 5). Also, the amount of the disease noted in the field alternately planted to beans and beets indicates that the wilt bacteria are capable of surviving in the soil for at least two winters between bean crops.

POSSIBLE REASONS FOR DIFFERENCES IN DISEASE INCIDENCE

Suppression of plant pathogens in soil is usually attributed to the activities of other soil-inhabiting microbes or to nutrient or environmental conditions directly unfavorable to the pathogens. Therefore an experiment was conducted to determine whether the difference in disease incidence in sandy loam and clay loam results from a difference in extent of microbial suppression of the bacterial wilt pathogens.

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Table 1. Bacterial wilt incidence on punched and nonpunched bean plants as related to soil type and cropping history.

Cropping history and field no.	Percent of punched plants showing wilt symptoms on		Percent of nonpunched plants showing wilt symptoms on	
	Clay loam	Sandy loam	Clay loam	Sandy loam
Field first year in beans[a]:				
1	0	15	0	1-5
2	0	37	trace	1-5
3	0	45[b]	trace	5-10[b]
4	0	17	0	1-5
5	-	25	-	1-5
Field second successive year in beans:				
1	0	20	0	1-5
2	0	20	0	1-5
3	-	50	-	1-5
Field third successive year in beans:				
1	5	25	0	10-20
2	7	-	0.5	-
Field alternately in beans and beets[c]:				
	5	-	0.5	-

[a] Not the first bean crop in the field, but the first since other unrelated crops.
[b] This field was planted with seed produced in an adjacent field the previous year. All other fields were planted with Idaho certified seed.
[c] Bean and sugar beet crops were alternated for 12 years. Sugar beets were grown in 1954.

Table 2. Bacterial wilt incidence on punctured pinto bean plants in inoculated sterilized and nonsterilized clay loam and sandy loam.

Concentration of inoculum[a]	Percent plants showing wilt symptoms on			
	Sterilized clay loam	Nonsterilized clay loam	Sterilized sandy loam	Nonsterilized sandy loam
1000	64	4	36	16
100	28	0	4	4
10	16	0	8	0
1	16	0	0	0
0	--	0	--	0

[a] A heavy suspension from the infected seeds was used as the 1000-part concentration. This was diluted with sterile water to give the lower concentrations indicated.

Inoculum of the orange variety of the wilt pathogen (C. flaccumfaciens var. aurantiacum) was prepared by grinding infected seeds in water in a Waring Blendor. The inoculum was strained through cheesecloth and added in various concentrations in equal volumes of water to pots containing sterilized and non-sterilized portions of the two soils. After 1 week pinto beans were planted and at emergence thinned to five plants per pot. Each plant was injured below the soil line as previously described. One month later the disease data summarized in Table 2 were recorded.

The inoculum was somewhat less effective in non-sterilized clay loam than in non-sterilized sandy loam. The reverse was true in sterilized soils. High concentrations of inoculum caused higher disease incidence than lower concentrations. Similar results were obtained in other experiments in which infected seeds were planted or pure cultures of the pathogen were added to the two soils.

These findings suggest that one factor responsible for a lower incidence of bacterial wilt in clay loam than in sandy loam is a greater suppressive action of the microflora upon the pathogen in the finer textured soil. In the absence of competitive microflora the conditions of the clay loam were even more favorable for the pathogen than were conditions in the sandy loam. From the reported ability of C. flaccumfaciens to remain viable for long periods in a desiccated condition (3), it seems reasonable that the more droughty nature of the sandy soil under field conditions might be another factor in this soil difference. Further investigation is needed for clarification of this point.

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FIELD EVALUATION OF ANTIBIOTICS FOR CONTROL OF CROWN GALL ON THE CULTIVATED HIGHBUSH BLUEBERRY[1]

Bert M. Zuckerman

Summary

Gall formation was not suppressed when crown-galled highbush blueberries were treated with antibiotics applied during an interval which encompassed dormancy through budbreak. Severe defoliation followed application of Acti-dione at 100 ppm. A significant reduction in yield was associated with all antibiotic treatments, with the exception of the neomycin treatment. In the three streptomycin-base treatments, as the concentration increased, the average berry weight decreased.

INTRODUCTION

In 1956 highbush blueberry bushes affected with crown gall, Agrobacterium tumefaciens (E. F. Sm. & Town.) Conn, were treated in tests to determine the effectiveness of several antibiotics in the control of this disease. Effects of the antibiotic treatments upon current year fruit production were observed throughout the experiment.

MATERIALS AND METHODS

The blueberry bushes treated in these tests were of the Cabot or Pioneer variety located in a field approximately 2 acres in area. They varied in height from 3 to 4 feet. About half of the Cabot and Pioneer bushes in the field were affected by crown gall.

Forty heavily infected bushes were selected for the test. Treatments were assigned to each bush by a random sampling procedure. The antibiotics used were 37% streptomycin sulfate (Agri-strep type-A), 15% streptomycin sulfate (Agri-mycin 100), 20% streptomycin nitrate (Phytomycin), Acti-dione, and neomycin sulfate[2].

Prior to spraying, five galls were carefully measured on each bush. Measurements to one-hundredth of a centimeter were taken of each gall at right angles and parallel to the stem. The five galls measured were sometimes found on one stem, and sometimes on as many as five separate stems. An average of 2.3 stems was tagged per bush. Stems that were tagged contained only clearly defined galls from the point where the tag was attached to the tips of the bush. Areas with evidence of gall eruptions that had not yet formed clearly defined galls were not tagged.

Antibiotics were applied at weekly intervals for 3 weeks starting May 9. The first spray was applied when the bushes were dormant, the second when some of the buds were swelling and breaking, and the third when most buds had broken. The spray was applied to the entire bush until dripoff. Approximately 250 ml of solution was applied to each treated bush. A hand sprayer was used to apply the sprays. All bushes in the field, including the controls, were sprayed twice with ferbam for mummy berry control, and dusted three times with 4% Malathion for the control of blueberry maggot.

The berries were picked twice, once on July 25 and for the second time July 30. The total production of ripe berries per bush at the time of picking is given in Table 2.

In March, 1957, 10 months after the first spray was applied, the labelled twigs were removed from each bush. Observations were made relative to the production of new galls and new gall tissue on each twig.

RESULTS

Acti-dione at 100 ppm induced severe defoliation; however, the plants produced new foliage

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[2] The author wishes to thank the following companies for supplying the antibiotics used in these tests: Merck & Co. (Agri-strep type-A), Chas. Pfizer & Co. (Agri-mycin 100), Olin Mathieson Chem. Corp. (Phytomycin), Upjohn Co. (Acti-dione), and S. B. Penick & Co. (Neomycin sulfate).

Table 1. Yields of crown gall affected blueberries sprayed three times with antibiotics compared to yield of unsprayed bushes.

Antibiotic	Concentration (ppm)	Replicate Bushes	Ave. Total No. Berries Each Bush[a]	Ave. Total Wt. Berries Each Bush[a] (gms.)	Ave. Wt. Each Berry[a] (gms.)
Streptomycin sulfate (37%)	400	4	11.7	9.6**	0.7
Streptomycin sulfate (37%)	1000	4	1.2	0.5**	0.3
Streptomycin sulfate (15%)	400	4	87.5	104.0*	0.8
Streptomycin sulfate (15%)	1000	4	6.5	3.4**	0.5
Streptomycin nitrate (20%)	400	4	37.5	35.9*	1.0
Streptomycin nitrate (20%)	1000	4	23.2	17.9*	0.7
Actidione	100	4	2.2	1.0**	0.3
Neomycin sulfate	1000	4	230.5	212.1	0.9
Control	---	8	307.7	256.1	0.8

[a]Averages computed on the basis of two pickings; only ripe berries picked.
* - significant at the 5% level.
** - significant at the 1% level.
Least difference for significance at the 5% level - 146.9.
Least difference for significance at the 1% level - 244.0.

after 6 weeks. Chlorosis was not associated with any of the treatments. A reddening of the leaves was observed on several bushes; however, no attempt was made to correlate this observation with the treatments.

Berry yields on a weight basis were significantly greater on control bushes than on all antibiotic treated bushes with the exception of the bushes treated with neomycin. Differences between yields of bushes sprayed with the three streptomycin-base formulations at 400 ppm are not easily explained. Some variation was attributed to one bush in the 15 percent concentration series, which yielded three times as much as the other replicates in this series. However, even if this source of variation were eliminated, there still appears to be a difference in yields, which could be attributed to the three different streptomycin formulations.

Although there were no significant differences in yield between the bushes treated with the streptomycin-base formulations at 400 ppm and those treated at 1000 ppm, there was a trend towards reduction of yield at the higher concentrations.

Berry size as affected by treatments was expressed as the average weight of each berry on the bushes included in the treatment. In the three streptomycin-base treatments, as concentration increased the average berry weight decreased.

When twigs were removed from the test bushes 10 months after the first antibiotic application, observations were made relative to the effectiveness of the treatments in control of crown gall. These observations are given in Table 2.

New galls (produced after antibiotic treatments) were covered with light brown bark, as opposed to the dark-brown coloration on old galls (produced the year before antibiotic treatments); consequently, new galls could be easily distinguished from old galls. A number of twigs contained areas in which new gall tissue had erupted but did not form clearly defined galls. Occasionally new gall tissue formed in areas immediately contiguous to old galls. Ob-

Table 2. Crown gall symptom expression on diseased Cabot and Pioneer blueberries before and after antibiotic treatments.

Antibiotic	Conc. (ppm)	No. Reps.	Galls Formed Before Treatments: No. Measured	Galls Formed Before Treatments: Average Size (cm)	Galls Formed After Treatments: No. Formed	Galls Formed After Treatments: Average Size (cm)	Other Symptoms After Treatments: Twig Dead	Other Symptoms After Treatments: Twig Almost Dead	Other Symptoms After Treatments: Newly Formed, Poorly Defined Gall Tissue[a]
Streptomycin sulfate (37%)	400	4	20	.65 X .58	6	1.13 X .59	4	1	6
Streptomycin sulfate (37%)	1000	4	20	.84 X .72	19	.75 X .64	6	0	2
Streptomycin sulfate (15%)	400	4	20	.83 X .72	37	.67 X .57	1	2	5
Streptomycin sulfate (15%)	1000	4	20	.61 X .52	5	.72 X .66	3	2	0
Streptomycin nitrate (20%)	400	4	20	.74 X .67	27	.78 X .65	4	0	4
Streptomycin nitrate (20%)	1000	4	20	.87 X .79	21	.78 X .62	0	0	6
Acti-dione	100	4	20	.80 X .67	16	.99 X .86	2	1	7
Neomycin sulfate	1000	4	20	.85 X .76	17	.76 X .63	0	2	4
Control	---	8	40	.80 X .70	29	.84 X .67	8	1	12

[a] Each general area in which poorly defined gall tissue was formed was tablulated as one occurrence.

servations of the number of occurrences of these conditions in each test series have been listed in Table 2 under the heading "Newly Formed, Poorly Defined Gall Tissue." Frequently the twigs on which the initial observations were made were dead or almost dead at the conclusion of the test. The number of occurrences of each of these conditions in each series is listed under the appropriate heading in Table 2.

In Table 2, the column "Number Formed" under "Galls Formed After Treatments" represents only clearly defined galls that were present in the areas tagged when old galls were measured at the beginning of the experiment.

Analysis of size and number of new galls that formed on treated and untreated bushes indicated that there was no significant decrease in size or number of new galls which could be attributed to the antibiotic treatments. The formation of new gall tissue was greater on treated bushes, in most cases, than on the controls. Where fewer new galls were produced, the reduction in number from those produced on the controls was not statistically significant.

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HOT-WATER TREATMENT OF AVOCADO SEED FOR THE ERADICATION OF PHYTOPHTHORA CINNAMOMI

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Avocado root rot, caused by Phytophthora cinnamomi Rands, has become the most important disease of this crop in California. The fungus, originally reported on avocado from Puerto Rico in 1927 (3), was first isolated from avocado roots in California in 1940 (4). Unofficial estimates at that time placed the number of affected acres at 500 (5); now some 2500-3500 acres, representing approximately 10 percent of the total avocado acreage in California, are involved (8). It is probable that this spread has been primarily due to movement of infected nursery stock (6, 7, 8).

While fumigation with methyl bromide or chloropicrin can free nursery soil of Phytophthora, the problem of eliminating the fungus from infected seed or nursery stock still remains. Attempts to do this by means of chemical drenches or dips have not in general been satisfactory. Preliminary experiments have shown that avocado seed and seedlings can withstand immersion in hot water at 120° F for 30 minutes (2). This fact, together with the relatively low thermal tolerance of Phytophthora spp. (1), indicated that a hot-water seed treatment might be an effective and inexpensive way of eradicating the fungus from infected plant parts.

A series of seed treatments using hot water at various temperatures and for various lengths of times was tested during 1956-57. Seed of both Mexican and Guatemalan varieties, which had been stored for various periods, was used. The results following a 30-minute treatment (Table 1) showed that the seed would tolerate a temperature of 125° F without any effect on seed germination. At 130° decreased germination was evident, while at 135° and above all seed was killed. At 120° hot-water treatment even for 1 1/2 hours did not reduce germination percentages. These results held true for both Guatemalan and Mexican varieties and for seed stored from 0 to 18 months.

Table 1. Germination of avocado seed after a 30-minute hot-water treatment at various temperatures. Counts were taken 2 months after planting.

Temperature °F	Number of seed	Number of seed germinating	Percent germination
68	20	18	90
120	122	118	97
125	59	55	93
130	35	18	51
135	40	0	0
140	40	0	0
No treatment	40	40	100

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Table 2. Germination counts and percent infection after a 30-minute hot-water treatment of infected avocado seed at various temperatures.

Temperature °F	Number of seed	Number of seed germinating	Percent infection
68	50	17	100
115	50	21	22
120	50	41	0
125	50	37	0
No treatment	50	23	100

Seeds which had been artificially inoculated with Phytophthora were also hot-water treated. Inoculations were made by introducing a small piece of mycelial mat into one cotyledon of each seed and incubating it in a moist chamber. After 2 to 4 weeks a firm, dark brown lesion on the seed was produced. The viability of the fungus both before and after the hot-water treatment was determined by culturing from the advancing margin of this necrotic area. The seeds were then planted in sterilized soil and after three months the seedlings examined and cultured if necessary. In some cases the fungus killed the embryonic growing points before the heat treatment so that the germination counts were not a reflection of the control achieved (Table 2). However, in no case was the fungus recovered from the 100 infected seeds which had been treated at either 120° F or 125° for 30 minutes. The plants from treated seed appeared healthy and showed no sign of root damage, even under periodic wet-soil conditions. All of the 50 infected seeds which were not heat treated showed evidence of fungus activity. Lower temperatures did not eliminate the fungus.

It has been recommended that since the Phytophthora fungus can enter windfallen fruit and grow into the seed, only seed which has been picked from the tree should be used for planting stock. Using seed treated with hot water at 120° to 125° F for 30 minutes eliminates the possibility of introducing the pathogen into the nursery on seed, even though it might have been originally infected. This treatment, coupled with a soil treatment, in the case of container-grown stock, or careful selection of a site when planting seed directly in the soil, should materially reduce the spread of the disease by means of infected nursery stock.

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SEVERE LOSS FROM STRAWBERRY LEAF SPOT IN ARKANSAS IN 1957[1]

J. L. Dale and J. P. Fulton[2]

Leaf spot of strawberry, incited by Mycosphaerella fragariae (Tul.) Lindau, caused unusually severe damage in many commercial strawberry plantings in Arkansas during the first week of May. In the northwest portion of the State severe infestations were observed, with some growers experiencing a total crop loss. Blakemore, which is the main commercial variety grown in Arkansas, is commonly affected with a moderate amount of leaf spot during summer and fall. However, early infection prior to and during harvest resulting in heavy fruit loss is unusual.

The outbreak of leaf spot was characterized by sudden appearance of symptoms, being reported simultaneously from central, north-central, and northwest Arkansas. In the central and north-central sections of the State infection was largely confined to leaflets and petioles. In these areas some beds had a scorched appearance from leaf infections, but there was little infection of fruit, fruit caps, or fruit stems. In some beds in the northwest part of the State all plant parts were affected, with 100 percent of the fruit showing "black seed" symptoms (Fig. 1). Even in instances where fruit was not infected, the market value of such berries was reduced because of spotting and death of fruit caps.

FIGURE 1. Symptoms typical of severe damage to leaves and fruit clusters affected by strawberry leaf spot. Note dead caps (calyxes) and fruit blemishes.

Continued and above average rainfall during April apparently was the main factor responsible for the leaf spot epiphytotic. Rainfall at the University Farm at Fayetteville, which was typical of the area of disease infestation, was recorded as 11.3 inches for the month. The normal rainfall for April is 4.7 inches. Approximately half the total rainfall occurred on three separate days during the first half of the month, while the remainder fell during the last 15 days, with rainfall being recorded for all but one day during the latter period.

The mean temperature for April was 57° F, compared with the normal mean of 59°. The mean for the first 15 days was 50°, and that for the last 15 days 65°. The mean high for the latter half of the month was 73°.

Observations revealed that in most instances there was little damage in plantings with good air and water drainage. Flat, poorly drained fields and sloping fields closely surrounded by woodlands showed greatest degree of injury. In general, plants with heavy foliage

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[1] Published with approval of the Director of the Arkansas Agricultural Experiment Station, Fayetteville, Arkansas.

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showed most severe infection. There was no detectable correlation between cleanliness of plants last fall, as far as leaf spot was concerned, and severity of infection this spring. There were no observations or reports of severe infection in fields where captan sprays or dusts had been applied regularly. This could of course be partially attributable to location of such plantings, and not entirely the result of effectiveness of the fungicide in controlling the disease.

ARKANSAS AGRICULTURAL EXTENSION SERVICE AND ARKANSAS AGRICULTURAL EXPERIMENT STATION, FAYETTEVILLE, ARKANSAS

RESOLUTION OF CERTAIN STRAWBERRY VIRUSES BY THE APHID VECTOR CAPITOPHORUS MINOR FORBES[1]

Forest Gay Rorie[2]

Summary

The aphid Capitophorus minor Forbes readily transmitted nonpersistent virus isolates from commercial strawberry plants to Fragaria vesca L. indicator plants. No evidence of transmission of persistent viruses was obtained.

Isolate I, a mild mottle virus, was studied in detail. The virus was acquired by the aphid in 1 hour but not in 30 minutes. It persisted in the aphids for a period of more than 2 but less than 3 hours. Symptoms are described in four different F. vesca indicators. Isolate II, with aphid-vector relationships similar to those of Isolate I, produced more severe symptoms in all 4 indicators.

INTRODUCTION

This investigation was undertaken to separate into their component parts some of the virus complexes that are common to strawberry plants in Arkansas. After the individual virus types were separated, their reactions in indicator plants were used to correlate the viruses in Arkansas with those in other areas. Capitophorus minor Forbes, the aphid most commonly found in Arkansas strawberry plantings, was the vector used in these studies. Information on the virus-vector relationships of this aphid is necessary to understand more fully the spread of strawberry viruses.

REVIEW OF LITERATURE

Since the literature on virus diseases of strawberry has recently been reviewed by Plakidas (7) reference will be made only to those publications specifically related to the work presented here. Several workers (6, 8, 9) have demonstrated that strawberry virus diseases are often caused by a complex of two or more distinct viruses which can be separated by an aphid vector. Certain of the viruses persisted in the aphid only a few hours and were termed "nonpersistent". Others persisted in the aphid for several weeks and were called the "persistent" components. Usually a virus complex caused more severe symptoms than either component alone.

Demaree and Marcus (2) described two virus complexes from Eastern United States which were aphid and graft transmissible. Two distinct types of symptoms, "Type 1" and "Type 2", were obtained in the indicator plant Fragaria vesca (East Malling Clone). McGrew (5) demonstrated that Type 1 symptoms were the result of a combination of mottle virus and strawberry latent virus, the latter having been previously described by Frazier (3).

Several aphid species have been reported as vectors of certain strawberry viruses (7). Demaree and Marcus (2) reported transmission of their Type 1 and Type 2 viruses by Capitophorus minor. Detailed studies of the virus-vector relationships of this aphid, however, have not been presented.

MATERIALS AND METHODS

Viruses were separated by allowing aphids to feed on infected plants of the Blakemore variety. After an acquisition feed, the aphids were transferred successively to a series of indicator plants. All transfers were made using a moistened camel's-hair brush.

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[2] The writer is indebted to Dr. J. P. Fulton for counsel and assistance in directing this study.

Sources of virus inoculum were from two field collections of Blakemore which had previously been indexed to the indicator plant Fragaria vesca and found to be infected. The indicators developed symptoms similar to those described by Demaree and Marcus (2) as Type 1 and as Type 1 plus Type 2. Clones from the original plants were maintained in the greenhouse.

To check for virus contamination, aphids (Capitophorus minor Forbes)[3] collected on wild strawberry plants were allowed to feed on the indicator plant F. vesca. Virus-free aphids were cultured on "virus-free" Blakemore plants enclosed in cages made of 32 x 32 mesh screen. The aphid colonies were kept in a temperature room at 21° C.

Four indicator plants of the wild species F. vesca, each showing a different virus reaction, were used. Two of these were from different sources of the East Malling Clone designated MEM and BEM[4]. The third indicator, designated as RAS, is a clonal line of a hybrid F. vesca[5]. The fourth indicator, designated AS, is a runnerless variety of the Alpine strawberry F. vesca[6].

Two methods were used for the aphid acquisition feeding periods. In one method aphids were colonized on an infected plant growing in soil and enclosed in a 32 x 32 mesh screen cage. The other method was to colonize aphids on a detached, infected leaf which was kept at high relative humidity in a moist chamber. The detached leaf petiole was inserted snugly into the constricted end of a vial made by pulling out 10-mm glass tubing. Grafting tape was wrapped around the petiole and the tube to prevent evaporation from the tube. After filling the vial with water, a small cork stopper was inserted in the open end. As transpiration occurred, water was replaced at the corked end of the tube. Aphids could be maintained on a leaf fixed in this manner for 3 weeks.

The infection feed was made by transferring aphids which had acquired the virus to indicator plants. Aphids were put in one of two types of cages for the infection feeds. One was a 32 x 32 mesh screen cage in which plants could be set. The other was similar to that described by Mellor and Fitzpatrick (6). The former type proved more satisfactory, especially for feeds of longer than 24 hours.

All acquisition and infection feeds were made in a 21° C constant temperature room. Following the infection feeds aphids were killed with nicotine sulfate spray and plants were moved to a greenhouse operated at a minimum temperature of 19.5° C.

The excised leaf grafting technique described by Bringhurst and Voth (1) was used in all grafting experiments.

EXPERIMENTS AND OBSERVATIONS

Two different nonpersistent virus isolates were separated from infected commercial plants by the vector Capitophorus minor. The isolates separated were designated as "Isolates I and II".

Aphid-Vector Relationships of Isolate I

Isolate I was acquired by C. minor after 1 hour in 4 out of 17 trials. In no instance out of 23 trials was the virus acquired in 30 minutes. Ten aphids were used in each of the acquisition experiments. This isolate was persistent for a period of more than 2 hours but less than 3 hours in C. minor. Aphids were given an acquisition feed of at least 24 hours on an infected Blakemore plant and then transferred to healthy indicator plants for an infection feed. After preliminary screening it appeared that the virus persisted in the aphids for a period of about 3 hours. The experiment was repeated. This time, aphids were given acquisition feeds on the infected source plants, and transferred to healthy plants at hourly intervals for 2 more hours. Virus was transmitted during the 2-hour infection feed and during the first 1-hour feed but not during the second 1-hour feed. Out of 32 trials no virus transmission occurred after 3 hours. No positive correlation was found between the length of the acquisition period and the length of the infection feed.

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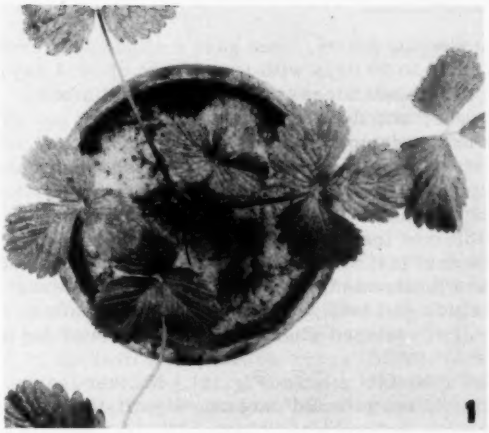
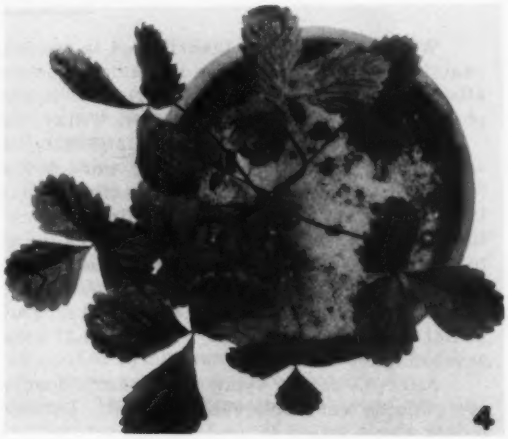
[3] Kindly identified by H. B. Boudreaux of Louisiana State University.

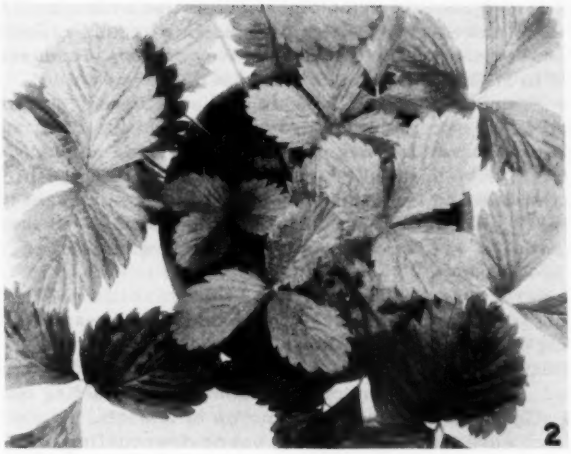
[4] The MEM clone was obtained from Michigan State University in 1952 and has been used at the University of Arkansas since that time. The BEM clone was obtained in 1956 from the United States Department of Agriculture Station at Beltsville, Maryland, where it had been used for a number of years as an indicator plant.

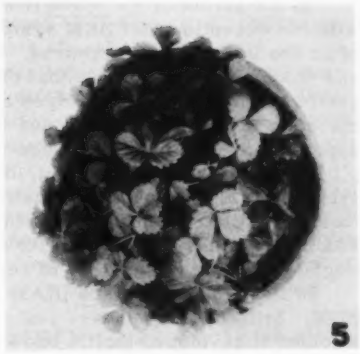
[5] Developed by N. W. Frazier (4) of California.

[6] Seed obtained as the variety Baron Solemacher.









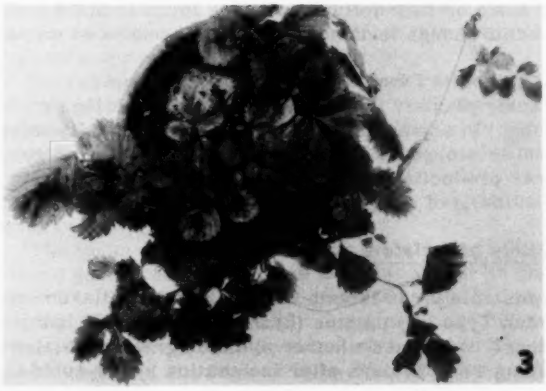


FIGURE 1, FIGURE 2. Isolate I: 1 -- in the Fragaria vesca indicator MEM; 2 -- in the F. vesca indicator RAS.

FIGURE 3, FIGURE 4. Isolate II: 3 -- in the F. vesca indicator BEM; 4 -- in the F. vesca indicator AS.

FIGURE 5. The heat stable component (virus C) in the Fragaria vesca indicator MEM.

Symptoms of Isolate I

When isolate I was transmitted to the four indicator plants, each gave a slightly different reaction. Initial symptoms normally appeared in 12 to 26 days with an average of 16.6 days after inoculation. Symptom expression in each of the indicators will be discussed in two phases: the initial symptoms, which may disappear, and the final symptoms.

In the indicator plant F. vesca (MEM), the initial symptoms on young leaves, if they were the first part to be affected, were downcurling, severe chlorosis, puckering, and malformation of one or more of the leaflets, usually near the edges. On leaves that were one-fourth grown to mature when the first "shock" of the disease became apparent, symptoms first appeared as severe downcurling of the petiole and leaflets and severe chlorosis along leaflet edges. One or more of the leaflets of the next leaf that unfolded following these initial symptoms usually became mottled, puckered, and malformed. Rarely did the later leaves develop malformation or necrotic areas. Downcurling of leaf petioles was not noted after initial symptoms appeared. Leaves that were fully developed when the shock occurred did not develop symptoms.

After the initial symptoms, plants developed a distinct mottle (Fig. 1). Neither leaves nor petioles were noticeably stunted. Runner length was affected only slightly, if at all. Infected plants failed to produce runners as readily as uninoculated plants. No bud proliferation occurred at any time, but leaf puckering occurred in a few cases.

In the indicator F. vesca (BEM) the development of symptoms was similar to that in MEM with the exception that BEM symptoms were more severe with more necrotic areas, especially after the initial shock appeared. One-fourth grown to mature BEM leaves developed chlorosis and downcurling similar to that observed in MEM. After the leaves that developed initial symptoms matured, their edges became irregularly serrated and ragged. Yellowed, puckered, chlorotic zones appeared around the necrotic areas, giving the leaves a distinctive, distorted appearance. As in MEM no downcurling of leaf petioles was noted after the initial shock.

Following the shock effect, the indicator developed mild Demaree and Marcus Type 1 symptoms (2). There was a distinct leaf mottle and the spots were slightly more discrete than those on MEM. Ruffling and puckering were evident in practically all of the leaves, but there was little, if any, stunting of leaves or petioles. Internode length of runners was reduced slightly, as was the number of runners produced.

In the hybrid F. vesca (RAS) the virus symptoms were less severe than in either MEM or BEM. Initially, new leaves just emerging from the crown developed epinasty. As each leaf unfolded it developed mottle and puckering of the lamina. In most cases, the unfolding leaf showed yellowing along the edge. Normally both the lamina and petiole of the leaf which showed initial symptoms were downcurled. The initial symptoms, in other than unfolding leaves, appeared as a downcurling followed by a slight yellowing and mild mottle of leaflets. Puckering of the tissue was common in leaves that were initially infected, but no downcurling or puckering appeared following initial symptoms. Leaves which unfolded after the initial symptoms appeared developed a mild mottle (Fig. 2). The chlorotic areas of the mottle were much less discrete in this plant than in either MEM or BEM. The edges of spots blended with surrounding healthy tissue rather than having more or less definite edges as found in MEM and BEM. Neither bud proliferation nor noticeable change in number of runners produced was noted. Leaflets and petioles were not stunted.

In the Alpine strawberry F. vesca (AS), Isolate I caused less severe symptoms than were produced in the other three indicator plants. Initial symptoms on unfolding or actively growing leaves appeared as mottling. In some cases leaves that were initially infected developed slight puckering. Infected leaves developed no downcurling or yellowing of edges. Observations on bud proliferation and runner production could not be made due to the plant growth habits. There was no appreciable stunting of either leaves or petioles.

Separation of Isolate II

The second nonpersistent aphid-transmissible virus isolate was carried in a Blakemore plant. It caused severe Demaree and Marcus Type 1 symptoms (2) in MEM and BEM indicator plants. Identical symptoms developed in these two clones whether aphid or graft inoculated. Symptoms developed in indicator plants during 11 to 24 days after inoculation by the aphid C. minor.

In preliminary experiments the virus did not persist in the aphid for a period of 24 hours, but was transmitted during a period of about 3 hours. The aphids acquired the virus during

a feeding period of 24 hours. Shorter acquisition periods were not investigated.

Symptoms of Isolate II

Isolate II virus caused a severe shock reaction in F. vesca (MEM). Leaves emerging from the crown became downcurled, chlorotic, mottled and developed small necrotic areas, later becoming crinkled. Often the entire leaf developed necrosis. Leaflets of older leaves developed severe downcurling, distortion, mottle, and chlorosis. The leaflets were somewhat elongated and puckered with irregular edges. Absence of one or more leaflet was common, especially after initial symptoms appeared.

Following the initial effects of the virus the bud became moderately proliferated, and most leaves were puckered. Leaves became mottled and extensive vein clearing developed, so that the entire vascular system was outlined when the leaf was held before a light. Runner production was reduced and plants on runners were relatively close together. Leaflets and petioles were about one-half normal size.

In the indicator plant F. vesca (BEM) the virus caused symptoms similar to those produced in MEM (Fig. 3). Generally, the only differences were more severe puckering and leaf necrosis that developed following initial symptoms. This symptom complex consisted of small necrotic areas, which dried and cracked, surrounded by zones of chlorosis. The leaflet edges were more irregular than those of MEM.

Isolate II caused milder symptoms in RAS than in MEM and BEM. Initial symptom expression consisted of relatively severe shock reaction manifested by downcurling, puckering or twisting, yellowing, mottle, and occasional necrosis of parts of leaflets.

Following the initial symptoms, leaves were puckered, mottled, and about one-half normal size. Leaflets elongated to a "feather-like" shape. The distinctive chlorotic vein clearing, as described in MEM, developed and slight bud proliferation occurred. Often only one side of a leaflet was malformed. Runner production was not significantly affected. No necrotic areas were noted after the initial symptoms appeared.

In AS, Isolate II (Fig. 4) caused little or no shock reaction. Initially, leaflets became downcurled, moderately chlorotic, and mottled. The next leaves to develop from the crown were puckered or ruffled, mottled, stunted to about one-half normal size, elongated, and often malformed on at least one side of the leaflets. Leaflets also developed chlorotic vein banding. The chlorotic areas of the mottle were not as discrete as those in the other three indicator plants.

When plant tissue infected with Isolate II was grafted to an MEM plant carrying Isolate I, the plant developed symptoms of Isolate II. Final symptoms were slightly more severe than those of Isolate II alone.

Persistent Component Transmission

Isolate II caused symptoms essentially the same as the graft transmitted virus from the original source. Isolate I, however, caused less severe symptoms in indicator plants than did the complex from which it was separated. Thirty-four attempts were made at aphid-transmission of a persistent component from this complex, but after infection feeds of at least 14 days, no transmission was obtained.

During the fall of 1955, aphids were collected from strawberry fields and transferred to healthy indicator plants. Twenty-five to 30 aphids were transferred to 3 each of the 4 indicator plants. After 1 hour and 24 hours they were retransferred to other healthy indicator plants. Symptoms of a mild to severe form of mottle similar to Isolate I appeared in every plant used in the 0-1 and 1-24 hour infection periods. After the 24-hour feed, aphids remained on the third set of indicator plants for 3 weeks. No virus symptoms later appeared in any of the plants.

The virus complex from which Isolate I was obtained produced symptoms in MEM similar to those described by Demaree and Marcus (2) as caused by the combination of "Type 1" and "Type 2". When an MEM plant infected with this complex was held 14 days in a heat chamber maintained at a minimum night temperature of 36° and maximum daily temperatures between 43° and 47° C the Isolate I portion of the virus complex was eliminated. The remainder produced symptoms similar to "Type 2" of Demaree and Marcus (2) or virus C of McGrew (5) (Fig. 5). Plants containing only heat-stable virus were grafted to plants infected only with Isolate I, and symptoms of the original complex were obtained.

In a further attempt to obtain evidence on the transmission of virus C, plots of virus-free

Blakemore plants were interplanted in the field with plants from a clone containing the complex. Aphids were noted on several occasions in this planting. After 1 year 73 plants from the virus-free plots were indexed by grafting to MEM. Nineteen plants developed mottle symptoms similar to those produced by Isolate I. No plants developed symptoms suggesting the presence of virus C.

DISCUSSION

Several workers have been successful in transmitting strawberry viruses with aphid vectors. Most workers used Capitophorus fragaefolii Ckll. and successfully transmitted the nonpersistent viruses. Similar results are presented in this study of the vector relationships of Capitophorus minor Forbes with viruses from Arkansas strawberry plantings. This aphid readily acquired and transmitted mild and severe forms of a nonpersistent virus designated "Isolates I and II".

Isolate I causes a mild mottle. It is essentially the same as mild strawberry mottle virus mentioned by Frazier (3). Isolate II is tentatively considered a strain of this same virus. The fact that there is no cross protection between Isolate I and II suggests, however, that the two strains are not closely related.

Virus 1 of Prentice and Harris (8) was apparently the same as those here designated Isolates I and II. Their description of Virus 1 as causing chlorotic speckling, distortion, and dwarfing of leaves corresponds with symptoms produced in this study by Isolates I and II when inoculated to plants of the East Malling Clones. The fact that Virus 1 was transmitted from acquisition feeds of 1 hour or more and persisted for 3 hours is more evidence that it is a similar virus. The nonpersistent virus component described by Mellor and Fitzpatrick (6) also showed symptoms similar to those produced by Isolates I and II. Their virus persisted in only one case for more than 4 1/2 hours and was acquired in 3 hours by the aphid C. fragaefolii.

Since there were different reactions with the nonpersistent isolates transmitted to all four of the indicator plants, there should be some attempt to explain the variations. The BEM plants were known to carry latent virus (strain A) and all symptom expression in this indicator plant must be considered as the symptoms of the latent virus plus a mottle virus. MEM plants probably also carry the latent virus or a strain of it. There was a marked difference in symptom expression between MEM and BEM in every case when Isolate I was present. MEM plants exhibited a more severe reaction to the isolates than did AS and RAS and, therefore, probably should be classified as carrying a latent virus. Frazier (3) has indicated strains of the latent virus.

Small necrotic areas surrounded by puckered chlorotic areas were observed on the leaves of most of the BEM plants when the plants were inoculated with Isolate I or II. These necrotic areas were believed to be one of the identifying characteristics of the latent virus when in combination with a mottle virus. Necrotic areas of this type were seen occasionally on MEM plants inoculated with any single isolate of mottle.

Further studies are necessary to determine the significance of the failure to obtain evidence of transmission of persistent components of the strawberry virus complex.

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APRICOT RING POX VIRUS NOT TRANSMITTED THROUGH APRICOT SEEDS

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Transmission of the ring spot virus through seeds of mazzard cherry (2), mahaleb cherry (1), and peach (3) raises the question whether other viruses affecting stone fruits are seed-transmissible.

Approximately 150 seeds from several ring-pox-affected trees of the Wenatchee (Wenatchee Moorpark) apricot variety were collected in Hemet, California in July 1952. The seeds were air-dried and stored dry until February 1953, when they were after-ripened by storage for 50 days in a mixture of moist peat and sand at 38° to 40° F. They were planted in the nursery row and 126 seedlings grew.

None of the seedlings have shown any virus or viruslike symptoms. It is known that certain varieties can be infected and maintain the ring pox virus without symptoms (4), therefore the 126 seedlings were indexed by budding from each into two fruiting-size Tilton apricot trees. No ring pox symptoms occurred on any of the index or check trees; therefore it seems conclusive that the ring pox virus is not seed-borne in apricots.

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WISTERIA MOSAIC AND PEONY LEAF CURL, TWO DISEASES OF ORNAMENTAL PLANTS CAUSED BY VIRUSES TRANSMISSIBLE BY GRAFTING BUT NOT BY SAP INOCULATION

Philip Brierley and Paul Lorentz[1]

Wisteria Mosaic: A mosaic in Wisteria sp., probably W. floribunda var. rosea, was submitted from a Michigan nursery in June 1955. When received the plants were leafless, but new leaves developed in our greenhouse showed diffuse yellowish blotches with scattered green islands of irregular size and shape. Mature leaves showed lateral twisting of the leaflets. W. sinensis inarched to the source plant in February 1956 developed the same symptoms in May, after 3 1/2 months' contact (Fig. 1). Similarly W. floribunda (hort. var. Koshigaya) from Glenn Dale, Maryland, inarched to the source plant in December 1955 developed yellow speckling and mottling in June 1956, 6 months after contact. Sap inoculation to wisteria, bean, tobacco, and Gomphrena was without effect. Myzus persicae failed to transmit the virus to bean or guar in a trial by R. V. Travis. A mosaic of W. sinensis was reported from New South Wales (2) in 1941, but no transmission tests were mentioned. Weiss (5) reported a mosaic of wisteria in Mississippi and New York.

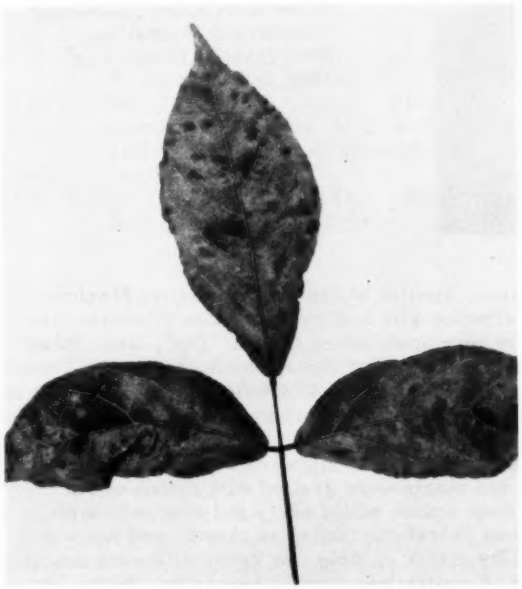

FIGURE 1. Symptoms of a mosaic in part of a leaf of Wisteria sinensis graft-infected February 10, 1956, and photographed June 15, 1956.

Peony Leaf Curl: A leaf curl of peony was brought to our attention by Dr. J. I. Hambelton of Brookeville, Maryland, in May 1954. Affected plants were about half normal height and had crooked flower stalks and conspicuously curled leaves. Flowers were normal in appearance, but unsalable as cut blooms because of their short stems. The disease was prevalent in the variety Mme. de Verneville at Brookeville, but not typically expressed in any other variety in the 1-acre planting. The source plants were kept in a coldframe over winter and returned to the greenhouse in April 1955. Regrowth in 1955 was weak and short, the shoots were thin, and there were no blooms and the leaves curled. In the field at Brookeville many plants of Mme. de Verneville were dwarfed and had crooked stems and curled leaves.

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FIGURE 2. Symptoms of peony leaf curl in Mme. de Verneville graft-infected February 27, 1956, and photographed October 22, 1956.

In May 1955, two plants from a local source, similar to the variety Festiva Maxima, were inarched to Mme. de Verneville plants affected with leaf curl, and one plant was root-grafted to these affected plants. No symptoms developed before October 1955, when these plants as well as a healthy stock of Mme. de Verneville from Ohio were stored at 40° F until February 1956. The plants grew rapidly on return to greenhouse conditions. The two plants inarched to the leaf-curl-affected plants developed strong leaf curl symptoms; the one plant root-grafted to the leaf-curl plant showed no effect.

In February 1956, two plants of healthy Mme. de Verneville were inarched to the plants infected by grafting the previous season, and two plants were grafted with scions of the original Mme. de Verneville source plants. Both scions wilted early and died before May. In March two more healthy plants were inarched to leaf-curl-affected plants, and one was grafted with a scion of such a plant. Ten healthy plants of Mme. de Verneville were inoculated by the carborundum leaf-rubbing method, and 10 contaminated by cutting knife. In the last test the knife was first passed through a young stem of a leaf-curl-affected plant and then used to make a diagonal cut in a young stem of a test plant; cuts were then closed and bound with raffia. No leaf curl appeared in any of these inoculated plants during that growing period.

All peonies were stored at 40° F in August 1956. After 5 1/2 weeks' storage, on September 12, 1956, they were returned to the greenhouse. Regrowth in autumn was prompt and normal; the first flowers appeared October 22. All four plants inarched to leaf-curl plants and two of three top-grafted with scions of such plants developed typical leaf curl symptoms (Fig. 2). The interval between inoculation and expression of symptoms, still from one growth period to the following one, was here shortened to 7 or 8 months. No leaf curl appeared in plants inoculated by leaf-rubbing or in those contaminated by knife. Inoculation by Yarwood's fresh-leaf method to Mme. de Verneville, tobacco, Gomphrena, sweet william, petunia, snapdragon, Silene compacta, and Verbesina encelioides was without effect. No virus was transmitted from infected peony to Vinca rosea by Cuscuta campestris.

Symptoms of peony leaf curl first expressed in grafted plants of Mme. de Verneville under glass are dwarfing, crooked stems, and curled leaves. Flower stalks on such plants

are about 30 percent shorter than those on normal plants and crooked as in field-grown infected plants. Flowers are normal in color and form. In the second and later years of infection plants are further dwarfed and have thin, weak, usually blind shoots. Leaf curl symptoms are less conspicuous and less consistently expressed when infections are long standing. Late stages of leaf curl, at least in greenhouse-grown plants, are not definitely distinguishable from other debilitating diseases of peonies.

The Brookeville planting is the only one known to us in which peony leaf curl has caused significant damage. Only one of several peony growers in other localities consulted was aware of this disease; he reported successful control by ploughing under the infected plants in his one experience with leaf curl. Since this disease is distinctive only in early stages, it is possibly more common than recognized. There was circumstantial evidence of natural spread at the Brookeville planting. Natural transmission of a virus of this type would imply vector activity, but no vector studies have been made.

Earlier records of peony diseases (1, 3, 4) may include references to the one we call leaf curl, but no transmission tests were reported.

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PERITHECIA OF ERYSIPHE POLYGONI ON ICELAND POPPY

Robert D. Raabe

Powdery mildew of poppies, though uncommon, has been reported from various countries throughout the world. Shaw (7) recorded Erysiphe polygoni DC. on opium poppy (Papaver somniferum L.) from India in 1918. In 1925 Zaprometoff (10) listed the opium poppy as being attacked by E. cichoracearum DC. in the Semiretchensk region of Russia. In his handbook on mildews in Russia, Jaczewski (5) listed a form of E. cichoracearum on P. somniferum and also on a Papaver sp. In addition, he listed a form of E. communis (Wallr.) Link on P. alpinum L. Blumer, in 1926 (1), recorded P. dubium L. as a host for E. polygoni and in his book in 1933 (2) he included P. rhoeas L., P. dubium var lecoquii and P. somniferum as hosts for E. communis. In 1935, Brundza (3) recorded E. cichoracearum on P. somniferum in Lithuania. The California poppy, Eschscholtzia californica Cham., was listed as a host for E. polygoni in California by Yarwood in 1937 (9). In 1945, Darpoux (4) recorded that in France P. somniferum var. nigrum was attacked by an Oidium which he presumed to be E. cichoracearum. According to Weiss' Index of Plant Diseases (8), E. polygoni has been found attacking P. somniferum in Oregon. According to these reports, perithecial formation was found only on the opium poppy, P. somniferum, and perithecia were recorded as those of E. cichoracearum by Brundza (3) and Jaczewski (5), and of E. communis by Blumer (2).

FIGURE 1. Non-infected leaf (left) and two powdery mildew-infected leaves of Iceland poppy.

Although there is no record of a powdery mildew on the Iceland poppy, P. nudicaule L., Kenneth F. Baker[1] has found the conidial stage of E. polygoni on this plant repeatedly since 1940 in the flower-seed-producing area near Lompoc, California. In the summer of 1956 a planting of Iceland poppy on the Berkeley campus of the University of California was found to be heavily infected with the conidial stage of a powdery mildew (Fig. 1). The conidial stage was of the E. communis type as shown by Blumer (2). This is the E. polygoni type of Salmon's classification (1). Infection was so severe that not only were many of the older leaves of infected plants killed but several plants died as a result of the infection. As the season progressed, the perithecial stage of the fungus was produced in abundance on the infected leaves (Fig. 2). The perithecia measured 85-115 μ in diameter and enclosed three to six slightly stalked asci measuring 38-45 x 61-73 μ. The asci contained 3 to 6 ascospores, most of which were about 12 x 22 μ. These measurements fall within the measurements as listed by Salmon (6) for the species E. polygoni.

[1] Personal Correspondence

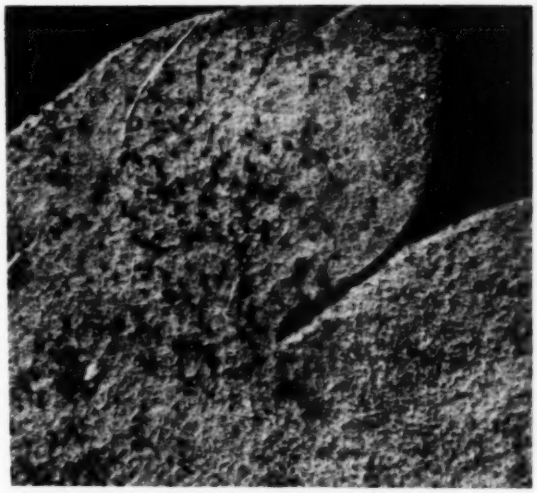

FIGURE 2. Close-up of Iceland poppy leaf showing perithecia of Erysiphe polygoni. The perithecia were produced on both surfaces of infected leaves.

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ROOT AND STEM ROT OF SOYBEAN CAUSED BY CORYNESPORA CASSIICOLA (BERK. & CURT.) WEI[1]

M. G. Boosalis and R. I. Hamilton[2]

Abstract

Corynespora cassiicola (Berk. & Curt.) Wei was shown to cause a heretofore undescribed root and stem disease of soybean (Glycine max (L.) Merril) in Nebraska. Dark, reddish-brown lesions which change to a dark, violet-brown color with age, and stunting of severely infected plants are characteristic symptoms of the disease. Most of the lesions observed on naturally infected plants were superficial and seldom extended beyond the first three parenchymatous cell layers of the cortex. Findings from field surveys and from soil temperature studies suggest that soil temperatures above 19° to 21° C arrest the development of the disease before it can damage the host sufficiently to affect appreciably the yield and/or the quality of the beans. The highest incidence of the disease was found in fields where soybeans had been planted for two successive years. The pathogen overwinters on infected root and stem tissues of soybeans and survives in infested, unsterilized soil for at least 2 years. Olive et al. (2) previously reported that Helminthosporium vignae Olive sp. nov. (Synonym Corynespora cassiicola) incites lesions on leaves of soybean and cowpea (Vigna sinensis (Torner) Savi). No symptoms of leaf infection were observed on soybeans grown in Nebraska.

Symptoms of an undescribed disease were observed in 1954 on the roots and on the transitional stem tissue of soybeans (Glycine max (L.) Merril) growing in several fields in the vicinity of Fremont, Nebraska. The seemingly threatening nature of this new disease prompted investigations to determine the disease syndrome, the causal agent, the disease cycle, and the potential economic threat of the malady.

SYMPTOMS

Field collections of soybean plants, of the varieties Hawkeye and Lincoln in the two-leaf stage demonstrated the presence of circular to ovular dark, reddish-brown lesions on the hypocotyl, on the tap roots and on the larger lateral roots. At this stage of development the lesions were approximately 2.5 mm in diameter. A girdling-type of lesion, like that seen on the lateral roots, was found infrequently on the hypocotyls and tap roots. A random distribution of several lesions commonly developed on a single lateral root whereas only one lesion was usually observed on the hypocotyls and tap roots. No symptoms or signs of the disease were manifested on the cotyledons and leaves of the plants.

With increasing age of the infected plants the lesions enlarged in size and in many cases caused girdling of the tap roots and adjacent stem tissue. Lesions on the lateral roots also elongated and often coalesced to cause discoloration of the entire root. With the onset of sporulation of the fungus pathogen, the lesions of all infected tissues changed from reddish-brown to a dark, violet-brown color. Severely infected plants were slightly stunted with no other symptoms developing on the aerial parts of the plant. The above disease syndrome was also produced on soybean plants of the varieties Hawkeye and Bansei grown in sterilized soil infested with the pathogen or with naturally infected roots and stems.

Although no symptoms of the disease were observed on leaves of soybean grown in the field, symptoms did develop on artificially inoculated leaves of plants grown in the greenhouse. Three-week-old soybean plants of the variety Bansei were sprayed with a conidial

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suspension of an isolate known to be pathogenic to soybean stems and roots. The plants were first placed in a moist chamber at 22° C for 24 hours prior to inoculation and were returned for a period of 36 hours after inoculation. After an incubation period of 5 days in a greenhouse maintained at 24° reddish-brown, circular to ovular lesions with a diameter ranging from 1 to 4 mm had developed on the inoculated leaves. Many of the spots were surrounded by a yellow halo approximately 1 mm wide. The leaf spot symptoms resulting from artificial inoculation closely resembled those incited by Helminthosporium vignae Olive sp. nov. (2). In addition to these leaf symptoms, a veinal necrosis and stem flecking were apparent. The fungus pathogen was easily isolated from the lesions on the leaves and stems either by the plate method or by placing infected tissues in a moist chamber and isolating the fungus conidia subsequently produced.

In a parallel experiment, leaves of cowpea (Vigna sinensis (Torner) Savi) of the variety Black Eye were inoculated in the same manner as were leaves of soybean. The pathogen incited reddish, purple spots on the leaves of cowpea. The lesions were considerably smaller than those formed on soybean leaves and they did not enlarge as did the lesions on soybean leaves. No annular rings were discerned within the leaf lesions as reported by Olive (2). Isolations from lesions on cowpea yielded the pathogen.

THE CAUSAL FUNGUS

The morphologic and cultural characteristics of the pathogen isolated from naturally infected soybean tissues were similar, except for minor differences of the conidium, to those of Helminthosporium vignae Olive (2) and to Corynespora cassiicola (Berk. & Curt.) Wei (3). Conidia harvested from two-week-old cultures of the fungus on potato-dextrose agar (PDA) measured 9.4-11.6 μ x 66-326 μ. When first formed the young conidium is hyaline to gray, becoming brown to black with age. The number of septa of mature conidia varied from 1 to 11. Germination of the asexual spores on PDA, pH 6.0, is primarily monopolar with bi-polar germination occurring frequently. Germination by either of these two modes occurs readily, in less than 24 hours, on PDA. After 36 hours germination of intercalary conidial cells may occur.

Nomenclature of the Fungus

An undescribed species of Helminthosporium was reported by Olive et al. in 1945 (2) as being a leaf spotting pathogen on cowpeas and soybeans in the southern United States. The fungus was subsequently named Helminthosporium vignae Olive (2). The nomenclature of this organism was revised in 1949 by Olive (1) who renamed it Helminthosporium vignicola Kawamura comb. nov. with Cercospora vignicola Kawam. and H. vignae Olive as synonyms. Wei (3) has recently reviewed the taxonomy of this fungus and has placed it in the genus Corynespora on the basis of the proliferation of a new conidiophore through the apical abscission layer of the existing conidiophore and because of the constantly terminal position of the conidia or chain of conidia on the conidiophore. Cercospora vignicola and H. vignae were reduced in this classification to synonymy with Corynespora cassiicola (Berk. & Curt.) Wei.

DISEASE CYCLE

The mode of overwintering and the longevity of survival of Corynespora cassiicola were studied by placing naturally infected soybean stems and roots on the surface of field soil contained in 8-inch pots which were buried outdoors in soil up to their brims. The survival of the pathogen was determined by making periodic isolations from the infected soybean tissue and by planting the highly susceptible soybean variety, Bansei, in the infested soil. It was found that the pathogen lives from one season to the next on the infected soybean stems and roots and that, moreover, the fungus can survive in infested, fallow soil for at least 2 years.

The initial infection of soybeans grown in the field occurred during the early seedling stage. Symptoms of the disease were observed on the underground parts of the host as early as 3 days after seedling emergence. Surveys of soybean fields for three consecutive years indicated that protracted periods of cool, moist weather during the early stage of plant growth were conducive to disease development. In this connection the cardinal temperatures for the pathogen on PDA, pH 6.5, are: minimum, 5° to 7° C; optimum, 18° to 21° C; maximum 34° to 39° C. Studies made in Wisconsin soil temperature tanks disclosed that 15° to 18° was the optimum temperature for infection and disease development on the soybean variety Hawkeye.

The 25-day-old plants harvested from infested soil kept at 15° to 18° were severely infected, with reddish-brown lesions running the entire length of the main root. Moreover, approximately 80 percent of the lateral roots showed infection. Compared to plants of the control treatment, the infected soybeans grown at 15° to 18° were markedly stunted. The ingress of the pathogen was considerably greater at 15° to 18° than at higher temperatures, with many lesions extending to the vascular system of plants grown at the lower temperature. This was in sharp contrast to the superficial lesions seldom extending beyond the third cell layers of the cortex, as observed with the majority of soybean plants grown in the field.

The optimum pH for growth of the fungus culture on a weight basis at 22° C for 20 days on Czapek's sucrose-nitrate solution was between 6.0 and 6.5.

ECONOMIC IMPORTANCE OF THE DISEASE

The writers are of the opinion that root and stem rot of soybean initiated by Corynespora cassiicola is of little economic importance in Nebraska. The striking, gaudy lesions on the infected underground parts of the host give a false impression. What appears to be a devastating disease is actually quite insignificant. It would appear that the onset of warm weather, which usually commences early in Nebraska's growing season, arrests the development of the disease, confining the pathogen to the surface cell layers of the cortex. The highest incidence and the severest development of the disease was found in those fields planted to soybeans for two successive years. It is possible that with continued cultivation of soybeans the prevalence of Corynespora stem and root disease may increase in the vicinity of Fremont, Nebraska. Even if the incidence of the disease does increase, it probably will not cause too much damage to the crop unless cool, moist weather prevails during the greater part of the spring and early summer.

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METHODS OF TESTING FOR DISEASE RESISTANCE IN THE CORN DISEASE NURSERIES AT ST. PAUL AND COMPARISONS OF 110 LINES OF CORN FOR RESISTANCE TO DISEASES IMPORTANT IN THE NORTH CENTRAL REGION[1]

J. E. DeVay, R. P. Covey, and D. B. Linden[2]

Cooperative tests between the Departments of Plant Pathology and Botany and Agronomy and Plant Genetics at the Institute of Agriculture, St. Paul, Minnesota are made each year to evaluate the resistance of certain lines of corn to pre-emergence or seedling rot, stalk rot, ear rot, northern leaf blight, and smut. Notes are also taken on other diseases which develop from natural infection. These tests are made primarily to provide comparative information on the disease resistance of inbred lines and various experimental crosses which may be of value to corn breeders in the North Central Region.

METHODS OF TESTING

Pre-emergence or Seedling Rot Nursery

Each line of corn is replicated at random three times, each replication consisting of 10 hills spaced at 1 foot intervals with two untreated seeds per hill. Rows are spaced 40 inches. Each hill is sublayered with approximately 100 cc of grain inoculum prepared according to the method of Schroeder, et al[3]. Inoculum consists of a uniform mixture of cultures of 8 to 10 virulent isolates of Gibberella zeae. The inoculum is covered with about 1/2 inch of soil and the seed is planted on top of this and covered with soil. A non-inoculated check plot is similarly planted except no inoculum or sterile grain mixture is used. Approximately 2 to 3 weeks after emergence, the seedlings in each row are counted. The resistance of each line of corn is evaluated on the basis of the difference between the averages of the number of seedlings in the untreated and treated replications. An emergence of at least 60 percent or 12 plants in the untreated rows is required for a valid test. If the difference is less than 4, the line is considered resistant; if between 4 and 6, it is moderately resistant; if between 7 and 9, it is moderately susceptible; and if the difference is 10 or more, it is considered susceptible.

Northern Leaf Blight, Smut, Stalk Rot, and Ear Rot Nurseries

In these combined nurseries each line is replicated at random three times, each replication consisting of 10 hills spaced at 1 foot intervals with one plant per hill.

Approximately 3 to 4 weeks after emergence, the plants are inoculated with Helminthosporium turcicum (northern leaf blight) and Ustilago maydis (smut). Corn leaves infected the previous year with H. turcicum are ground in a Wiley mill to #20 mesh size and about 5 cc amounts of this material are sprinkled into the leaf whorls according to the method of Elliott and Jenkins[4]. This inoculation procedure is repeated 1 and 2 weeks later. Smut spores (1,000,000 viable spores per ml), suspended in tap water containing 0.1 percent Tween 20, are applied with a knapsack sprayer. The plants are sprayed until drops of water begin to run into the leaf sheaths. The plants are treated in the same manner 1 and 2 weeks later. Notes on disease reaction of the lines of corn tested are taken 2 to 3 weeks after silking for both northern leaf blight and smut.

The reactions of plants to northern leaf blight are judged as follows: If there are approximately 1 to 2 lesions (less than 1 inch in length) per leaf the line of corn is considered resistant; if 10 to 25 percent of the leaf area is affected, it is moderately resistant; if 26 to 60 percent of the leaf area is affected, it is moderately susceptible; and if more than 60 percent of the leaf area of the plant is affected, it is susceptible.

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[1] Paper No. 3792, Scientific Journal Series, Minnesota Agricultural Experiment Station.

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Table 1. Evaluation of disease resistance of lines of corn tested in 1956 under conditions at St. Paul, Minnesota[a].

Entry No.	Pedigree	Preemergence or seedling rot	Northern leaf blight	Smut				Stalk rot		Ear rot	
				Leaf	Stalk	Tassel	Ear	Gibberella	Diplodia	Gibberella	Diplodia
1	Minhybrid 511	MR	R	R	R	MR	R	MR	S	R	S
2	(Oh51A x M14) x Il-23	R	R	R	R	R	MR	MR	S	R	S
3	(Oh51A x M14) x Il-66	R	MR	R	R	MS	R	MR	MS	R	S
4	(Oh51A x M14) x Ml-1	R	R	R	R	R	R	R	S	R	S
5	$Wf9^T$ x Oh51A	R	R	R	R	R	R	R	MS	R	S
6	($Wf9^T$ x Oh51A) x W10	R	MR	R	R	R	R	R	S	R	S
7	" x B14	R	R	R	R	R	R	MR	S	R	S
8	" x M14	R	MR	MR	R	R	R	MR	S	R	S
9	" x H19	R	R	R	MS	R	R	MR	S	R	S
10	" x W22	R	MR	R	R	R	R	R	MS	R	S
11	" x Oh43	MR	R	R	R	R	R	R	MS	R	S
12	" x A73	MR	MR	MS	R	R	R	R	MS	R	S
13	" x A223	MR	R	MR	R	R	R	R	MS	R	S
14	" x A239	MR	R	R	R	MR	R	MR	S	R	S
15	" x A266	R	R	MR	MR	R	R	R	MS	R	S
16	($Wf9^T$ x Oh51A) x A277	MR	R	MR	MR	R	R	MR	S	R	S
17	" x A286	MR	MR	MS	R	R	R	MR	S	R	S
18	" x A289	R	R	R	MR	R	R	R	MS	R	S
19	" x A295	R	R	R	R	R	R	MS	MS	R	S
20	($Wf9^T$ x Oh51A) x A545	MR	MR	MS	R	R	R	R	MS	R	S
21	B14 x M14	R	MR	R	R	R	R	MR	S	R	S
22	A73 x A334	MR	MS	MS	MS	R	R	MR	MS	R	S
23	A286 x A295	MR	MR	R	R	MR	R	MR	S	R	S
24	A264 x A265	MR	MR	S	MS	MR	R	R	S	R	S
25	Minhybrid 508	R	MR	R	R	MR	R	R	MS	R	S
26	A286	MR	MS	MR	R	R	R	MR	S	R	S
27	A250	MR	MR	R	R	R	R	MR	S	R	S
28	A251	MS	MR	R	R	R	R	R	MR	MR	S
29	A254	R	MR	R	R	R	R	R	MS	R	S
30	A255	MR	R	MS	MS	R	R	MR	S	MR	S
31	A264	MR	MS	R	R	R	R	R	MS	R	S
32	A266	MR	MR	R	R	MR	R	MR	MS	R	S
33	A277	MR	R	R	MR	R	R	MS	S	MR	S
34	A279	R	MR	R	R	R	R	R	MS	MR	S
35	A287	MR	MR	R	MR	S	R	MS	S	S	S
36	A288	MR	R	R	R	R	R	MR	S	R	S
37	A289	MR	R	R	MR	S	R	MR	S	MR	S
38	A293	MR	R	R	R	R	R	MR	S	R	S
39	A294	MR	R	R	MR	R	R	MS	S	R	S
40	A295	MR	R	R	MS	MS	R	MR	MS	MR	S
41	A296	MS	MS	R	R	MS	R	MR	MS	MR	S
42	A297	MR	MS	R	R	R	R	MS	S	MR	S
43	A401	S	R	MR	R	R	R	MS	S	MR	S
44	A402	MR	MR	R	R	R	R	MS	S	MR	S
45	A403	MS	R	R	R	R	R	MS	S	MR	S
46	B2	MR	MR	MR	R	R	MR	R	R	MR	-
47	Ia 153	MS	R	R	S	MR	R	MS	S	R	S
48	Wf 9	R	R	MR	R	R	R	MR	S	R	S
49	Oh 5	MR	MR	MR	MS	R	R	MR	S	R	S
50	M14	MR	MR	MS	R	MR	R	MS	S	R	S
51	W22	R	MS	MS	S	R	R	R	MR	MS	S
52	Oh 43	MS	MR	MR	MR	R	R	MR	S	MR	S
53	Oh 51A	MR	R	R	R	MR	R	MR	MS	R	S
54	A71	MS	MR	R	R	R	R	MR	S	MR	S
55	A73	MR	MS	R	R	R	R	MR	MS	R	S
56	A223	MR	MR	R	R	R	MR	MR	S	R	S
57	A322	S	MR	R	MS	MS	R	MR	MR	R	S
58	A334	R	MS	R	R	S	R	R	S	R	S
59	A340	MS	MR	R	S	R	R	R	MS	MS	S
60	A344	MR	R	R	MR	R	R	MR	S	R	S

Table 1. (Concluded)

Entry No.	Pedigree	Preemergence or seedling rot	Northern leaf blight	Smut				Stalk rot		Ear rot	
				Leaf	Stalk	Tassel	Ear	Gibberella	Diplodia	Gibberella	Diplodia
61	A357	MR	MS	R	MR	R	R	MR	S	R	S
62	A374	MS	MR	R	MR	R	R	R	S	R	S
63	A375	MR	MR	R	R	R	R	MR	S	MR	S
64	A385	MS	MR	R	R	MR	R	MR	S	MR	S
65	A392	MS	MR	R	R	R	R	MR	S	R	S
66	Os 420	MR	MR	R	MS	R	R	MR	S	R	S
67	B9A	S	MR	R	R	MS	R	MR	MS	MR	S
68	W10	S	MR	MR	MR	R	R	MS	S	MR	S
69	A434	MR	MS	R	R	R	R	MR	MS	R	S
70	(A434 x W22) F5[c]	MR	MS	R	MR	R	R	R	MR	MR	S
71	"	MR	MS	R	MS	R	MR	R	MR	MR	S
72	"	R	MS	R	MS	R	R	R	MS	MR	S
73	(A434 x W22) F5[c]	MR	MS	R	MS	S	R	R	R	MS	S
74	"	R	MS	R	MS	S	R	MR	MR	MR	S
75	"	MR	MS	R	MS	R	R	MR	MS	R	S
76	"	MS	MR	R	MR	R	R	R	MR	MR	S
77	"	MR	MR	R	MR	R	R	MR	MR	MR	S
78	"	R	MS	R	S	MS	MR	MR	MR	R	S
79	A34	R	MS	R	R	R	R	MR	S	R	S
80	(A34 x W22) $W22_1S_3$[c]	R	MS	MS	S	R	R	R	MR	MR	S
81	"	MR	MR	MS	R	R	R	R	R	MS	S
82	"	MS	MS	MR	MS	R	MS	R	MR	R	S
83	"	R	MS	MR	S	R	R	R	MS	MR	S
84	"	MS	S	R	MR	R	R	R	R	R	S
85	"	R	MR	R	MS	MS	MS	R	R	MR	S
86	A171	MS	MS	R	R	R	R	MR	S	R	S
87	(A171 x Oh 43) F5[c]	MR	MR	R	R	R	MR	MR	S	R	S
88	"	MS	MS	R	MR	R	R	MR	S	R	S
89	"	MR	MR	R	R	MR	R	MR	MS	R	S
90	(A171 x Oh 43_1S_3[c]	MR	MR	MS	MS	MR	R	MR	S	MR	S
91	"	MR	R	R	MR	MS	R	MR	MS	R	S
92	"	MR	R	R	R	R	R	MR	S	R	S
93	A166	S	MR	S	R	MR	R	MR	MS	MR	S
94	(A166 x A277) F5[c]	MR	MR	R	R	MS	R	MS	S	R	S
95	"	MR	R	R	R	S	R	MR	MR	R	S
96	"	MS	MR	R	R	MR	R	MS	S	R	S
97	"	MR	MR	R	R	MR	R	MR	MS	R	S
98	(A166 x A277)$A277_1S_3$[c]	MS	MR	R	R	R	R	MS	MS	R	S
99	"	MR	R	R	R	R	R	MR	S	MR	S
100	A216	MR	MR	R	R	MR	R	MR	S	MR	S
101	(V3 x Wf9) F4[c]	MR	MS	R	MS	R	R	MR	S	R	S
102	"	MR	MS	R	MS	MR	R	MR	S	R	S
103	V3	MS	MS	R	S	MS	R	MS	S	MR	MS
104	A321	MR	MR	R	R	R	R	S	S	MR	S
105	(V3 x A321)$A321_1S_2$[c]	S	MR	R	R	R	R	MS	S	MR	S
106	"	MS	MR	R	R	R	R	MS	S	MR	S
107	"	S	MR	MR	R	R	R	MS	S	MR	S
108	"	S	MR	R	R	R	R	MS	S	R	S
109	"	MR	R	R	R	R	R	S	S	R	S
110	"	MS	R	R	R	R	R	MS	S	R	S

[a] S - susceptible
MS - moderately susceptible
MR - moderately resistant
R - resistant

[b] Gibberella zeae and Diplodia zeae, respectively.

[c] Lines having the same pedigree are different selections from the same cross.

The reactions of plants to leaf, stalk, tassel, or ear smut are judged as follows: if 7 percent or less of the total number of plants treated have galls at a particular location, the line of corn is considered resistant; if between 8 and 13 percent, it is moderately resistant; if between 14 and 23 percent, it is moderately susceptible; and if the amount is 24 percent or greater, it is susceptible.

Stalk rot inoculations, using toothpicks according to the method of Young[5], are made about 7 days after silking. The second and fourth extended internodes (6 to 8 inches in length) above the ground are used for stalk rot tests. Picks are inserted in the internodes, midway between the nodes. Two to 3 weeks later the stalks are split open and the extent of the discolored areas is recorded. If less than 25 percent of the internode is discolored, the line of corn is considered resistant; if approximately 26 to 50 percent is discolored, it is moderately resistant; if approximately 51 to 75 percent is discolored, it is moderately susceptible; and if the discolored area extends the full length of the internode or into the internodes above or below, it is susceptible.

Ears are inoculated using toothpick inoculum[5] when the grain is in the milk to soft dough stage. Picks are inserted midway between the ends of the ears. Notes are taken 1 to 2 weeks after inoculation. If 25 percent of the ear is rotted, the line is considered resistant; if 26 to 50 percent is rotted it is moderately resistant; if 51 to 75 percent is rotted it is moderately susceptible; and if more than 75 percent of the ear is rotted, it is susceptible.

In all disease nurseries susceptible checks are included to measure the validity of the tests. The results given in Table 1, although only representing data from 1956, are of particular value in that excellent epidemics of the various diseases were obtained and comparisons in performance of the different lines of corn can be made.

DEPARTMENT OF PLANT PATHOLOGY AND BOTANY AND DEPARTMENT OF AGRONOMY AND PLANT GENETICS, INSTITUTE OF AGRICULTURE, UNIVERSITY OF MINNESOTA, ST. PAUL, MINNESOTA

[5] Young, H. C., Jr. 1943. The toothpick method of inoculating corn for ear and stalk rots. Phytopathology 33: 16.

PLANT PARASITIC NEMATODE GENERA FOUND IN THE VICINITY OF SMALL GRAIN ROOTS IN GEORGIA[1]

Luther L. Farrar

The Georgia small grain crop usually exceeds 1,000,000 acres annually. More than half of this is planted to oats. During the 1956-57 growing season a survey was made to determine parasitic nematode genera associated with small grains. From November to March a total of 87 samples was collected from 49 counties scattered throughout the State. These samples were from 67 oat fields, 13 wheat fields, one rye field, and one barley field, and five small grain nurseries.

PROCEDURE

Samples were taken with a 16-inch "New Long Core Soil Sampler", inserted at approximately a 45-degree angle under and with the drill rows. Several cores were taken at random from each field, composited, and thoroughly mixed. A 100-ml beaker aliquot from each field sample was processed, using a 325-mesh screen and the Baermann funnel technique.

RESULTS

Parasitic nematodes were observed in each sample. Pratylenchus sp. was most frequently observed, with Helicotylenchus sp., Trichodorus sp., Xiphenema sp., Tylenchorhynchus sp., Rotylenchus sp., Criconemoides sp., Meloidogyne sp., and Hopolaimus sp. following, in that order. It was not unusual to observe 300 to 400 Pratylenchus sp., and frequently as many as 1750, per pint of soil. Helicotylenchus sp. usually did not exceed 500; however, in one field 1435 per pint of soil were observed. Xiphenema sp. seldom exceeded 200 per pint of soil. The other genera usually numbered less than 100 per pint of soil.

It should be noted that no pathogenicity studies have been conducted as yet. Genera observed are presented in Table 1.

Table 1. Parasitic nematode genera found in Georgia small grain fields, and number of fields infested[a].

Nematode genera	Number of fields infested			
	Oats	Wheat	Rye, barley, and small grain nurseries	Total
Pratylenchus sp.	56	10	5	71
Helicotylenchus sp.	29	4	4	37
Trichodorus sp.	28	3	1	32
Xiphenema sp.	20	4	3	27
Tylenchorhynchus sp.	14	3	3	20
Rotylenchus sp.	3	1	1	5
Meloidogyne sp.	2	0	0	2
Criconemoides sp.	2	1	0	3
Hopolaimus sp.	2	0	0	2

[a] Number of fields sampled: oats 67; wheat 13; rye 1, barley1, small grain nurseries 5; total 87.

DISCUSSION

Since large populations of potentially pathogenic nematodes have been found in the vicinity of small grain roots in Georgia, field, greenhouse and laboratory studies are planned on identity and pathogenicity of the species present, soil fumigation, varietal responses, and the effect of trap crops and summer fallow upon nematode populations. To attain these objectives, fields with high populations have been selected for field plot studies and pure populations of some genera have been established for greenhouse and certain basic studies.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

1 Contribution of Department of Plant Pathology, Georgia Experiment Station. Journal Series No. 319.

SCREENING MISSISSIPPI SOILS FOR PLANT PARASITIC NEMATODES

G. K. Parris[1]

Abstract

The cause of decline of turf grass, ornamentals, cotton, and pecan in Mississippi is demonstrated to be often due to attack by plant endo- and ectoparasitic nematodes including Tylenchorhynchus spp., Trichodorus spp., Rotylenchus spp., Criconemoides spp., Hoplolaimus spp., Xiphinema spp., Paratylenchus spp., and Pratylenchus spp.

INTRODUCTION

Since July 1955, the author has been engaged in attempting to ascertain the possible nematological cause of poor growth or the death of established turf grass in Mississippi. A few other plants, mostly ornamentals, whose poor growth or death has puzzled growers, also have been studied from this standpoint. The soil was screened and processed according to the Christie-Perry modification of the Baermann technique[2]. Soil collections were made, where possible, from healthy-appearing turf, from affected turf, and from the margin between these two areas. The nematodes collected were very kindly identified by Dr. J. M. Good, Associate Nematologist, Agricultural Research Service, United States Department of Agriculture, Nematology Field Laboratory, Tifton, Georgia, who made recommendations as to whether or not the numbers of plant parasitic nematodes found were sufficient to consider them a likely etiological agent of injuries noted. This paper reports the results of findings to April, 1957.

RESULTS

Lawns

Twenty-one lawns, 18 of St. Augustine grass (Stenotaphrum secundatum), 2 of Zoysia grass (Zoysia spp.), and 1 of Centipede grass (Eremochloa ophiuroides) have been analysed for possible nematode attack. In 9 lawns plant parasitic nematodes were considered to be active etiological agents of yellowing or death of the affected areas of the lawns. Six of these lawns were of St. Augustine, 2 of Zoysia, and 1 of Centipede.

In 9 of the remaining 12 lawns, plant parasitic nematodes were found to be present, but their abundance was probably not sufficient to incriminate them specifically as causal agents of disease. In 3 lawns no plant parasitic nematodes were found. Relative frequencies of the plant parasitic types of nematodes were as follows: stylet (stunt) nematodes (Tylenchorhynchus spp.) 8 lawns; stubby root nematodes (Trichodorus spp.) 6 lawns; spiral nematodes (Rotylenchus spp.) and ring nematodes (Criconemoides spp.) 5 lawns each; lance nematodes (Hoplolaimus spp.) 4 lawns; dagger nematodes (Xiphinema spp.) 3 lawns; pin nematodes (Paratylenchus spp.) 2 lawns; and meadow nematodes (Pratylenchus spp.) 1 lawn.

Towns and instances where lawns have been studied are Laurel (5), Jackson (3), Greenwood (2), with Pass Christian, Osyka, Columbus, Biloxi, Philadelphia, Monticello, Belzoni, and Louisville one instance each.

Ornamentals

Thirteen soil areas, involving Azalea (6), Boxwood (4), Camellia (3), Cedar (1), and Begonia (1), that showed poor growth were sampled. In 50 percent of the cases with azalea and boxwood respectively, 33 percent of the cases with camellia, and 100 percent with cedar and begonia, plant parasitic nematodes were considered as significant and causes of plant decline.

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[1] Plant Pathologist, State Plant Board of Mississippi and Head, Botany Department, Mississippi State College, State College, Mississippi.

[2] Christie, J. R. and V. G. Perry. 1951. Removing nematodes from soil. Proc. Helm. Soc. Washington 18(2): 106-108.

With azalea, nematodes found causing decline were stylet or stunt (5 instances), stubby root (1), spiral (2), and dagger (1); with boxwood, stubby root (1), stunt (1), lance (1), spiral (2), dagger (3), and meadow (1); with cedar, stubby root (1); with begonia, spiral (1); and with Camellia, stubby root (1), and spiral (2). Towns and instances where these samplings were gathered were Cleveland (3), with Hattiesburg, Hillsboro, Egypt, Starkville, Laurel, Columbus, Lucedale, and Natchez one instance each.

Other Plants

One soil sample each from stunted corn (Starkville), dying cotton (Tunica), and chlorotic pecan (Gulfport) has been studied. Even though aerial stunting and root symptoms on the corn were typically those of attack by stubby root nematodes (Trichodorus spp.), no significant numbers of these nematodes, or of any other plant parasitic nematode, were present. The cotton was shown to be suffering from attack by lance, tobacco stunt (Tylenchorhynchus claytoni), dagger, and stubby root nematodes, while the pecan was suspected of being injured by spiral and ring nematodes.

CONTROL

Recommendations were made to growers to use soil fumigants (Nemagon, V-C 13 Nemacide, etc.) where the need seemed practical. On lawns where nematodes could not be established as causal agents of decline, the application of dieldrin (2 to 3 pounds of the technical product per acre) has given good recovery of the turf in several instances. Local entomologists are unable to explain the degree of control obtained by dieldrin since no insect pests can be established to be present.

MISSISSIPPI STATE PLANT BOARD AND MISSISSIPPI STATE COLLEGE, STATE COLLEGE, MISSISSIPPI

BRENTIDS MAY BE VECTORS OF THE OAK WILT DISEASE[1]

W. D. Buchanan[2]

The Brentid (Arrhenodes minuta[3] (Drury)) is one of a very few insects that are known to: (a) visit oak wilt fungus mats caused by Ceratocystis fagacearum (Bretz) Hunt, (b) visit fresh wounds on oak trees, and (c) make holes in the exposed xylem of living trees for oviposition. These habits suggest that A. minuta may have a role in the spread of the oak wilt disease.

A. minuta has not been observed on the relatively few oak wilt fungus mats found in Missouri. However, in May 1955, E. B. Himelick of the Illinois Natural History Survey and the writer found considerable numbers of them on oak wilt fungus mats on several trees in the Sinissippi State Forest in northern Illinois. This beetle, although not specifically attracted to mats, will feed upon cultures of the oak wilt fungus and ingest the small globules of exudate that often form on the surfaces of the fungus. A. minuta is attracted to and hides in natural crevices in the bark and also in cracks such as those that develop in the bark over oak wilt fungus mats.

In the 4-year period 1953-1956, more than 2400 black, scarlet, and white oaks were blazed, or wounded in other ways, in oak wilt vector studies in Missouri. In observations that were made on these trees, and on hundreds of others, it was found that A. minuta was attracted to a very high percentage of fresh blazes made in May and June, and to a lesser degree to blazes made at other times. Although these insects will not bore through the bark, they do bore into exposed xylem, and the females oviposit in holes which they bore into the xylem. The eggs hatch into larvae that bore deep into the xylem where they may come in contact with C. fagacearum if the tree has oak wilt. Adults that develop from such larvae could have the fungus on or in their bodies, and carry it to healthy trees.

In 1954, 257 adult A. minuta were collected from blazes made on healthy black and scarlet oaks. These insects were divided into two lots. The first lot of 151 beetles was placed directly into cages that enclosed freshly made blazes on six healthy black oaks. The second lot of 106 beetles was held for 24 hours in Petri dishes containing well-developed cultures of the oak wilt fungus. The insects were then placed in cages that enclosed freshly made blazes on four healthy black oaks. None of the six trees exposed to the first lot of 151 beetles developed symptoms of oak wilt. But one of the four black oaks exposed to the second lot of 106 beetles did develop typical symptoms, although the oak wilt fungus could not be recovered from any of several samples that were taken from the symptomatic tree. However, several investigators agreed that the symptoms were typical for the disease. Much difficulty was encountered in attempting to isolate the causal fungus from other trees that developed typical oak wilt symptoms in Missouri in 1954; this may have been due to the unusually high summer temperatures which prevailed in that year.

In 1956 only 31 A. minuta were collected from wounds on healthy trees. All 31 of the beetles were held for 24 hours in Petri dishes containing well-developed cultures of an albino strain of the oak wilt fungus. Twenty-four of the beetles were then placed in cages on four healthy black oaks, one of which was wounded. Oak wilt developed in the wounded tree and the albino strain of the fungus was recovered[4]. No symptoms developed in the three trees that were not wounded. The other seven beetles were placed in cages that enclosed fresh blazes made on two healthy 7-foot red oak nursery seedlings. Oak wilt developed in one of them, and the albino strain of the fungus was recovered.

The beetles in the above tests were liberated several inches from the blazes, but it was only a matter of minutes until most of them had found the blazes. Once they had gotten on a blaze they remained there. In some instances they crawled directly to the blazes. No tests

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[1] Contribution from the Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, and the Missouri Agricultural Experiment Station, Journal Series No. 1764. Approved by Director.

[2] Forest Entomologist, Central States Forest Experiment Station, Forest Service, United States Department of Agriculture, Columbia, Missouri.

[3] Coleoptera - Brentidae.

[4] All of the cultures and identifications of the oak wilt fungus were made by Dr. T. W. Bretz, Professor, Forestry Department, University of Missouri.

were run in Missouri with A. minuta taken from oak wilt fungus mats because mats have never been found in Missouri from late April to mid-July which is the time when the insect is in the adult stage. However, it seems logical to believe that A. minuta would be as likely to become contaminated by contact with natural fungus mats as by contact with Petri dish cultures of the fungus. The above tests indicate that A. minuta may be a vector of the oak wilt disease.

CENTRAL STATES FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND THE MISSOURI AGRICULTURAL EXPERIMENT STATION

ACTI-DIONE TREATMENT OF BLISTER RUST TRUNK CANKERS ON WESTERN WHITE PINE

Virgil D. Moss[1]

Summary

In the development of a damage-preventive measure to preserve adequate stocking in western white pine stands that become infected by blister rust before Ribes are destroyed, Acti-dione was effective in killing mycelium in the perimeter of excise trunk canker wounds. Because of the short distance Acti-dione penetrates bark from the margin of wounds, the cut surface should extend above mycelial extent in the distal end of cankers and to the edge or slightly beyond the lower and lateral outer limits of surface discoloration. To avoid weakening trees by girdling too much bark from around a trunk, bark removed to expose mycelium should not exceed half the circumference of a trunk at any one point. Bark at the edge or slightly beyond the lateral outer limits of surface discoloration should be longitudinally slit to expose mycelium when the margin of a canker extends beyond half the circumference of a trunk. Acti-dione in oil is generously applied to the cut surface of wounds to complete treatment. Endomycin and calcium sulfamate are not effective in this type of treatment.

Few diseases of trees afford better opportunities for direct control than white pine blister rust, Cronartium ribicola Fischer. First, the alternate host plants (Ribes, currants and gooseberries) can be destroyed to prevent the rust from infecting white pines. Second, trees that become infected can be saved by pruning diseased branches before mycelium advances into the trunk, or after it enters the trunk, usually by removing enough bark to contain the fungus (3).

The best opportunity to save infected white pines comes when a diseased branch is pruned before mycelium advances into the trunk. At this stage no special operating skill or knowledge of the rust is required to obtain satisfactory results. Applied in its simplest form to take advantage of the preponderance of cankers located in the lower branch whorls, all lower branches are pruned to about one-third the crown height of a tree (4). After mycelium enters the trunk, technical skill and knowledge are essential to excise a high percentage of cankers successfully. Stewart (6), studying methods to eliminate cankers from young eastern white pine, Pinus strobus L., reports saving 100 percent by pruning the diseased branches before mycelium entered the trunk as compared with a minimum of 69 percent by cutting bark out of the trunk from about stubs of pruned diseased branches where the proximal margin of the branch canker was within 6 inches of the trunk.

Success of the excise trunk canker method depends upon whether adequate allowance is made in bark removal for mycelial extent beyond the outer limits of surface discoloration. Ehrlich and Opie (1), studying the longitudinal development of the rust on western white pine, P. monticola Dougl., found that surface discoloration bears no constant relationship to mycelial extent as the distance it extends beyond canker margin is variously increased by the age of canker, stage of canker, and thickness of bark at the outer limits of surface discoloration. Accordingly in practice a distance is arbitrarily fixed so the amount of bark removed from the trunk beyond the outer limits of surface discoloration supposedly is in excess of maximum mycelial extent for each pine species. Martin and Gravatt (3) allow for mycelial extent by cutting out bark 1 1/2 to 2 inches laterally and 3 to 4 inches longitudinally beyond the outer limits of surface discoloration on eastern white pine, and approximately doubling each of these distances to compensate for the faster growth of the rust on western white pine and sugar pine, P. lambertiana Dougl. They found that bark can be removed laterally to about half the circumference of a trunk without seriously weakening a tree.

Concomitant with the highly successful methods developed for Ribes eradication (5), equally effective damage-preventive measures are needed to preserve adequate stocking of white pine

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[1] Forester, Development and Improvement Unit, Division of Blister Rust Control, Region One, Forest Service, United States Department of Agriculture.

FIGURE 1. Western white pine excised blister rust trunk canker 3 years after treatment by Acti-dione. Surface discoloration shows how mycelium grows in a lateral and upward direction above wounds to appear as an arrowhead. Lower and lateral margins of wound are callusing where mycelium was killed by Acti-dione. Trees in full sun often are sunscald by pruning lower branch whorls in late spring and during the hot summer months.

FIGURE 2. Wood-rotting fungi present when dead bark was removed to prepare blister rust trunk canker for treatment by Acti-dione have not reinfected cut surface of wound that is coated by pitch. Neither have wounds been attacked by bark beetles or wood-boring insects.

when young forest stands become seriously infected by the rust. This paper reports a preliminary study to improve the excise trunk canker method by applying an antifungal substance to cut surfaces to kill mycelium in the perimeter of wounds.

MATERIALS AND METHODS

The antifungal substances, Acti-dione[2] (cycloheximide), endomycin[3], and calcium sulfamate[4], were applied to the cut surface of wounds of excise trunk cankers on 20-year-old western white pine in a plantation in the Coeur d'Alene National Forest, Idaho. Trees were selected for treatment if a trunk canker was producing pycniospores or aeciospores and the rust was unlikely to girdle the trunk before completing the tests. All structural stages of the rust were treated when more than the one canker was found on a trunk. As many as four trunk cankers were treated on a single tree. Each treatment was applied to 10 trees during late July in 1953.

Acti-dione and endomycin were applied to the cut surface of wounds at concentrations of 150, 300, and 600 ppm. Acti-dione was dissolved in 5 ml of acetone and the solute applied in the isoparaffinic base oil S/V Sovaspray 100[5]. Endomycin was dissolved in 5 ml of methanol before adding the solute to water containing 1% Triton B-1956[6] as a wetting agent. Calcium sulfamate (5%) was tested in the following 3 forms:

Form A	Form B	Form C
10 gr calcium sulfamate	50 gr calcium sulfamate	10 gr calcium sulfamate
10 ml glycerol	200 ml water	100 ml water
126 ml isopropyl alcohol	600 ml isopropyl alcohol	80 ml isopropyl alcohol
14 ml pine oil	150 ml glycerol	10 ml nonic (Sharples)
10 ml Tween #20	50 ml Tween #20	10 ml glycerol

Lower limbs from about one-third the crown height of trees first were pruned by shears and saw to eliminate branch cankers and facilitate working close to the trunk in canker treatment. A hatchet and pocket knife were used to remove bark from inside the margin of surface discoloration to 1) expose mycelium, 2) destroy insect infestations, and 3) eliminate areas of necrotic tissue that hinder callus formation and are a medium for infection by wood-rotting fungi.

The amount of bark that was cut from a trunk varied with the age, stage, and size of canker. Cankers in the aeciospore stage, because they contain a large amount of dead and dying bark and are covered by pitch, required the removal of the most bark from the trunk to expose mycelium. Pycniospore cankers were more easily prepared by removing the small area of bark containing pockets of varying sizes and depths of dead and dying tissue from about the base of the branch by which infection entered the trunk. A small amount of live bark from juvenile cankers was removed to expose mycelium and facilitate penetration by the antifungal substance.

The mass of dead and dying bark from aeciospore and pycniospore cankers first was cut out by a hatchet. A knife was used to clean remnants of bark from the xylem and trim by sloping the edges of the wound. Juvenile cankers were prepared by removing with a knife a small ring of bark from about the base or off the short stub of the pruned branch from which infection entered the trunk. The antifungal substances were generously applied to the cut surface of wounds with a small paint brush.

RESULTS AND DISCUSSION

Excise trunk canker wounds were examined for mycelial activity each year after treatment for 3 years. Bark about wounds was observed for 1) pycniospore and aeciospore pro-

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2, 3 Acti-dione and endomycin were kindly supplied for these tests by William Klomparens, The Upjohn Company, Kalamazoo, Michigan.

4 Calcium sulfamate was furnished by S. S. Sharp, E. I. DuPont de Nemours and Company, Wilmington, Delaware.

5 S/V Sovaspray 100 was supplied by W. B. Stevens, Socony-Vacuum Oil Company, New York, New York.

6 Triton B-1956 was supplied by F. J. Ampthor, Rohm & Haas Co., Philadelphia, Pennsylvania.

Table 1. Effect of Acti-dione, endomycin, and calcium sulfamate on blister rust, Cronartium ribicola, as shown by mycelial activity in the perimeter of excise trunk canker wounds on western white pine trees. Dates of treatments and results are July 1953 and October 1956, respectively.

Treatment	Cankers treated - number				Cankers killed - percent			
	J	P	A	Total	J	P	A	Total
Acti-dione (cycloheximide) (ppm)								
150	6	3	9	18	100	67	78	83
300	7	5	10	22	86	80	70	77
600	8	3	9	20	100	33	78	80
Endomycin (ppm)								
150	4	5	5	14	25	20	0	14
300	8	4	6	18	63	75	0	44
600	10	5	7	22	50	40	0	36
Calcium sulfamate (5 percent)								
Form A	4	3	9	16	50	0	0	13
Form B	5	2	10	17	40	50	10	24
Form C	6	5	8	19	33	20	0	16
Check cankers	5	5	7	17	60	20	0	24

J = juvenile canker; P = pycniospore stage; A = aeciospore stage.

duction, 2) swollen bark, color and extension of surface discoloration, 3) formation and growth of callus tissue, and 4) parasitism by the purple mold fungus, Tuberculina maxima Rostrup.

Structural stages of the 183 cankers in all treatments included 63 juvenile, 40 pycnial, and 80 aecial, or percentages of 34, 22, and 44, respectively. Pycniospore and aeciospore cankers usually produced one or the other spore stages 1 year after treatment if there was no inhibitory effect from the antifungal substance. Mycelial activity in juvenile cankers was determined by the increase in swelling of the infected bark tissue and the orange-yellow color of surface discoloration. For cankers in all structural stages of the rust, callus tissue was greatly retarded or entirely inhibited by active mycelium in any portion of the bark about wounds. The presence of the purple mold fungus, which attacks only the pycnial and aecial structures of the rust (2), often furnished additional proof of mycelial activity. Final results from macroscopically examining the cankers each year after treatment for the 3 years are presented in Table 1.

Check trees contained a total of 17 trunk cankers of which 4 were killed by the bark removal operation. Three of the 4 were juvenile cankers, 1 a pycnial canker, and all on a single tree. Two causal factors suspected in killing these cankers were 1) bark drying back from the edges of the wounds or 2) resistance inherent in this particular tree. The first obviously was not responsible because only the lip or exposed edge of the wounds dried. The fact that this tree far exceeded all others in the development of callus tissue probably accounts for the inhibitory effect upon mycelium.

Acti-dione concentrations of 150, 300, and 600 ppm were about equally effective in killing all structural stages of the rust. The 60 cankers treated by this antibiotic include 21 juvenile, 11 pycnial, and 28 aecial. Three years after treatment live cankers consisted of 1 juvenile,

4 pycnial, and 7 aecial, or percentages of 5, 36, and 25, respectively. These 12 live cankers are divided among concentrations as follows: 150 ppm, 3 cankers; 300 ppm, 5 cankers; and 600 ppm, 4 cankers; or based upon the number treated by each concentration, percentages of 17, 23, and 20, respectively. Accordingly, the lowest level of Acti-dione in these tests was lethal to mycelium in bark about excise trunk canker wounds.

Eleven of the cankers survived treatment by Acti-dione because the cut surface in the distal end of cankers was not extended to bring mycelial extent within range of the short distance the antibiotic penetrates bark above wounds. Although all cankers of this type did not survive treatment, the 11 cankers show that the cut surface of wounds should extend beyond the outer limit of surface discoloration in the distal end of cankers to facilitate penetration by the antibiotic. The extra amount of bark required to extend a wound a few inches beyond surface discoloration will not add to weakening a tree. An interesting phenomenon for this type of canker is the way mycelium has grown in an upward and lateral direction above wounds to appear as an arrowhead (Figure 1). No causal factor was determined in this study to explain the reason mycelium has not grown downward to reinfect bark tissue from which it was killed by Acti-dione.

The distance of penetration by Acti-dione was greatest in a downward and lateral direction from the border of wounds. Mycelium in the lower and lateral margins in all trunk canker wounds was killed by the antibiotic with the exception of one canker. Acti-dione, in this single canker, failed to move laterally around the trunk to kill mycelium that had grown beyond the base of a branch in the same whorl and close to the one by which infection entered the trunk. This near branch was pruned but its stub was not treated by Acti-dione. Unless there is danger in weakening a tree by girdling too much bark from around a trunk, the cut surface should extend to the edge or slightly beyond the lower and lateral outer limits of surface discoloration to assure penetration. If surface discoloration exceeds half the circumference of a trunk, bark at the edge or slightly beyond the outer limits of surface discoloration can be slit along the longitudinal axis of the trunk to avoid weakening a tree by removing too much bark from around a trunk.

Wood-rotting fungi in evidence when dead bark was removed to prepare cankers for treatment by Acti-dione appear to have been killed by the antibiotic. The surface of wounds, coated by a layer of pitch, has not become infected by fungi during the 3-year period following treatment (Figure 2). Neither have wounds become infested by bark beetles or wood-boring insects after treatment. However, if cankers are excised in the seasonal period insects are in flight, it would be advisable to incorporate an insecticide in the Acti-dione oil formulation to guard against attack by injurious insects.

Endomycin and calcium sulfamate were not effective in the treatment of excise trunk canker wounds. Calcium sulfamate, at a 5 percent concentration, was phytotoxic to western white pine. Its upward translocation was traceable by the moderate to severe burning of needles on branches originating directly above the excise trunk canker wound. This injury often extended to the top of many trees. Acti-dione and endomycin were not phytotoxic to foliage of western white pine when applied to the cut surface of excise trunk canker wounds.

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BOOK REVIEW

"BIOLOGY AND CONTROL OF THE SMUT FUNGI", by George William Fischer and Charles Stewart Holton. x + 622 pages. 1957. Ronald Press Company, New York, N.Y. Price $10.00.

Biology and Control of the Smut Fungi is probably the most comprehensive book ever published on this subject. Fischer's The Smut Fungi and Manual of the North American Smut Fungi, which could be termed companion volumes, are culminated in this work which in every sense is the last word on the smut situation. The Smut Fungi (1951) is a compilation of the literature on biology and control of some 330 species of smuts and the diseases they cause and serves as a guide to an extensive bibliography. The Manual of the North American Smut Fungi (1953), on the other hand, is a taxonomic handbook for the identification of all North American smut fungi that had been recognized and classified up to that time. It contains a Host Genus Index and keys to the species and to the genera of the smuts, with the species concept being based largely on morphology.

In this new book Fischer and Holton have encompassed the entire field. They have given new meaning to previously published material which is still tenable and have supplemented this with hitherto subordinated phases of the smut problem, such as control practices and the economic importance, both directly and indirectly, of this group of fungi to man. In addition, the number of smut species has been more than tripled. The bibliography occupies almost 150 pages.

Chapter 1 is an introduction to morphology, taxonomy and symptomatology of the smuts. The authors discuss families, genera and species, emphasizing the lack of agreement among authorities as to a uniform system of taxonomy, due chiefly to a divergence of opinion as to whether morphology or host specialization should be the foundation for classification. According to their own species concept based principally on morphology but recognizing host specialization at the family level, Fischer and Holton have set forth a Host Family Register of the Ustilaginales of more than 1100 species in 33 genera, the species being catalogued by host family. Of course there are other authorities who disagree absolutely with this system, considering that the host family is too broad a classification basis for species, preferring instead to employ the generic level. This chapter also contains a Key to the Genera of the Ustilaginales and a Synopsis of the Genera.

Chapter 2 deals with the history and economic importance of smuts. The first section is a searching historical review of the very earliest work down to present-day endeavors. The second section, on economic importance, is a factual accounting of the enormous devastation suffered by the cereal crops of the world. When one considers that "the cereals constitute 73 percent of the total food consumed by man" and that annual wheat loss in the United States due to bunt alone is counted in millions of dollars, one begins to realize the impact of this fungal group on the economy of a nation and to appreciate the statement that "probably the smuts are second only to the rusts in importance as diseases of cereal crops". Losses in other countries are given whenever the data were available.

Nomenclature and phylogeny are discussed briefly in the next chapter.

Chapters 4 through 10 cover the broad term "biology". The first, on secondary effects on the host, Chapter 4, is divided into the two categories of morphological and physiological modifications. Each type of secondary effect is treated separately, with specific examples and sources cited wherever possible. It is interesting to learn that there is at least one case on record where smut infection has resulted in a decreased susceptibility to powdery mildew: apparently the bunt Tilletia caries inhibits the development of Erysiphe graminis on wheat.

Chapter 5 investigates modes of infection, namely, seedling, embryo, shoot, and local, and relates the history of the discovery of each. Certain morphological aspects of spore development and germination are taken up. There is a thorough debate on the sexual nature of smut spores, -- a controversy that has raged since the days of De Bary and Brefeld. Other factors affecting parasitism are also treated.

Chapter 6 is concerned with the morphological variation expressed by the smuts on different agar culture media. In the next chapter, on cytology, the three main nuclear phases of the smuts are examined in detail and the importance of nuclear phenomena in relation to species delimitation is considered. This chapter also reports on the current thinking in regard to conjugate division and formation of clamp connections, and discloses the fact that chromosome research in the smuts is rather meager, principally because of the difficulty of gaining a true picture of meiosis from the relatively tiny nuclei. Chapter 8 follows logically with hybridization, mutation and genetics, which, as the authors state, constitute another comparatively new

area of investigation.

Chapter 9 is a dissertation on physiologic specialization, with stress on the host or pathogenic specialization of various smut species, since economically this is the most important of the race differential criteria and is common to all species.

Chapter 10 informs the reader of the great range of variability in reaction of host species and varieties to different races of smuts. Despite the fact that the subject of resistant varieties has been extensively studied and that new resistant types are constantly being developed, nevertheless the nature of resistance apparently is still anybody's guess; evidence basing resistance on cell-sap acidity, hydrogen-ion concentration, osmotic pressure and/or the seed germination process is so conflicting that this is still a virgin field of exploration. And, in spite of the discouraging capacity of rust races for adapting themselves to new host germ plasm, the possibility of finding truly resistant stable varieties is always present as long as continued effort is expended toward the discovery of specific resistance factors or genes and methods of inheritance in relation to known race populations of the smut species involved. When that time arrives, the "hit or miss" method of determining resistant varieties will be a thing of the past and the "vicious circle" that now exists in applying the use of host resistant varieties as a control practice will be eliminated.

The effects of smuts on man and animals makes an intensely absorbing Chapter 11. A number of cases are cited of the occurrence of respiratory allergies and other minor ailments afflicting human beings, and a wealth of evidence, both pro and con, as to the deleterious effects of smuts on farm animals, with most of the evidence seeming to support the negative view. The use as vegetables of shoot smut of wild rice in Formosa, China and Japan and of the young smut galls of corn in Mexico is of interest.

Methods and techniques for spore germination, pure culture studies, host inoculation, identifying and classifying physiologic races, cytological and histological studies, and testing fungicides for smut control, are described in Chapter 12.

The final and very possibly the most important chapter is on control practices other than the use of resistant varieties. Here are discussed the relative merits of various chemicals such as copper compounds, organic mercury compounds, formalin, and the polychlorobenzenes in seed treatment, the efficiency of hot-water treatment in control of loose smuts, the treatment of soil with fungicides to control soil-inhabiting smuts (onion smut in particular), the use of antibiotics such as Acti-dione against bunt of wheat and covered smut of oats, and cultural practices such as tillage, seeding dates, crop rotation, eradication of diseased plants or infected parts, and the value of fertilizers.

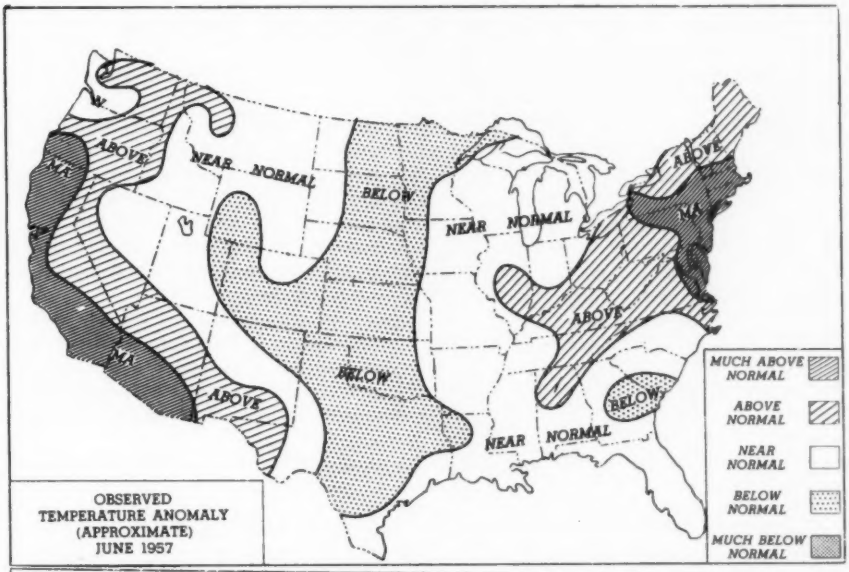
Although a large portion of the material in this book is based on conditions affecting smut development in the Pacific Northwest and other major wheat producing regions of this country, still whenever the statistics were available the authors quoted figures from other countries on crop losses, reports on occurrence, and so forth. There are more than 100 informative figures and nine tables.

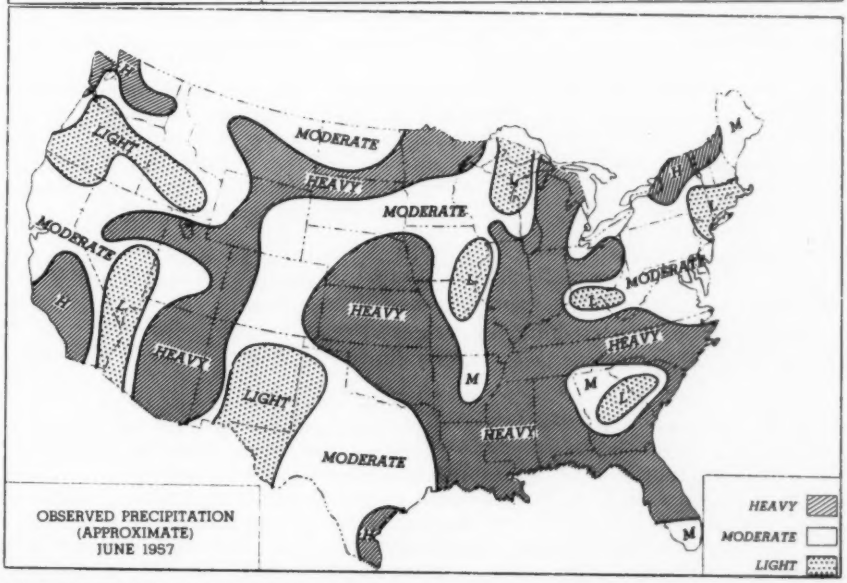
The book achieves a well-rounded exposition of knowledge and theory concerning the smuts. It is highly recommended for both experienced workers and beginners in smut research, as well as for any other persons who may be curious about smuts.

In view of the rapidly changing situation in the study of the fungi as a whole and of the smuts in particular it would appear that the last line quotation (page 449), concerning the need for up-to-date information, is particularly apt. -- HILDE McGRATH

CORRECTION

REPORTER for May 15 (Vol. 41, No. 5), page 384. Initial for senior author's first name is L instead of D. -- (L. J. Tyler).





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

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(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

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(10) The MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- for Model 1250. Most of the Experiment Stations have this type of multilith machine.

(11) We are the official correspondents in the United States of the PLANT PROTECTION BULLETIN published monthly by the United Nations Food and Agriculture Organization. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our monthly summary reports. Please mark it clearly so that we will know it is intended for the Bulletin. (See PDR 36: 492; 37: 637) -- P. R. M.